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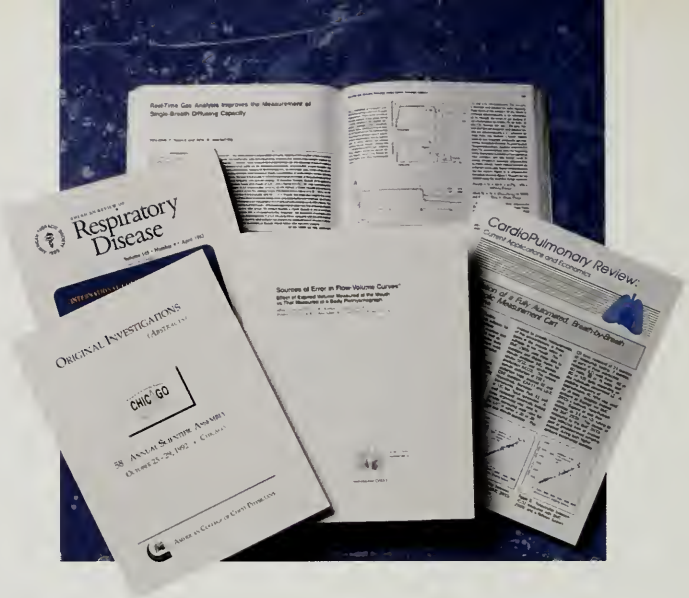

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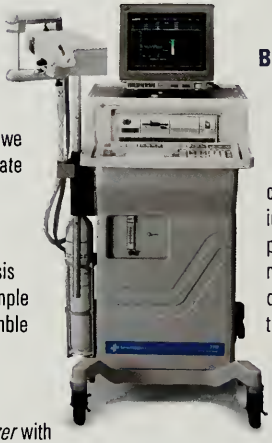

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Can We Predict Mortality for Low Birth Weight Infants? (editorial)—AT Costarino, RC Raphaely. Crit Care Med 1993;21:2. (Pertains to Horbar et al paper abstracted on Page 440.)

A New Therapy for the Adult Respiratory Distress Syndrome (editorial)—RC Bone. N Engl J Med 1993;328:431. (Pertains to Rossaint et al paper abstracted below.)

Pandemonium in the Modern Hospital (sounding board)—GW Grumet. N Engl J Med (Feb 11) 1993;328:433.

Surfactant and the Adult Respiratory Distress Syndrome (state-of-the-art review)—JF Lewis, AH Jobe. Am Rev Respir Dis 1993;147:218-233.

Neuromuscular Blockade in the Intensive Care Unit: More than We Bargained for (clinical commentary)—J Hansen-Flaschen, J Cowen, EC Raps. Am Rev Respir Dis 1993;147:234-236.

Performance Evaluation of Three Vaporizing Humidifiers and Two Heat and Moisture Exchangers in Patients with Minute Ventilation > 10 L/min—C Martin, L Papazian, G Perrin, P Bantz, F Gouin. Chest 1992;102:1347.

STUDY OBJECTIVE: To compare the thermal and humidification capacity of three heated hot water systems (HHWSs) and two heat and moisture exchangers (HMEs) in ICU patients submitted to minute ventilation > 10 L/min. DESIGN: Prospective, controlled, randomized, not blinded study. SETTING: ICU of a university hospital. PATIENTS: ICU patients requiring controlled mechanical ventilation with minute ventilation > 10 L/min. Patients had to be sedated and paralyzed and had to require ventilation for more than 4 days. INTERVENTIONS: Following a randomized order, the patients were ventilated for 24-h periods with three HHWSs (Bennett Cascade-2 humidifier, Fisher-Paykel MR460 and MR600) and two HMEs (Pall Ultipor and Hygrobac filter). MEASUREMENTS & RESULTS: In each patient and for each 24-h period, absolute humidity (AH), relative humidity (RH) of inspired gases, and tracheal temperature were obtained. Two HHWSs (Bennett and Fisher-Paykel MR460) had a better thermal and humidification capacity than any other systems ($p < 0.001$). The hydrophobic HME (Pall filter) had a poor thermal and humidification capacity (RH: 79 ± 8.7 %; AH: 20.6 ± 2.3 mg H_2O/L). The hygroscopic filter (Hygrobac filter) had better thermal and humidification capacity than the Pall filter (RH: 92.5 ± 3.6 %; AH: 29.1 ± 1.8 mg H_2O/L; $p < 0.001$). Tracheal temperature was well preserved by all systems. The thermic and humidification capability of the Hygrobac filter declined over 24 h. Since the Pall filter could not achieve an AH > 25 mg H_2O/L in any patient, it was not studied beyond the first measurement. CONCLUSIONS: The Hygrobac filter had a thermal and humidification capability close to the two HHWSs (81 to 97%) but the capability declines over 24 h. The Pall filter had a poor capability (54 to 74% of that of HHWSs).

Inhaled Nitric Oxide for the Adult Respiratory Distress Syndrome—R Rossaint, KJ Falke, F López, K Slama, U Pison, WM Zapol. N Engl J Med 1993;328:399.

BACKGROUND: The adult respiratory distress syndrome (ARDS) is characterized by pulmonary hypertension and right-to-left shunting of

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Rescue

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CONTRAINDICATIONS

None known.

WARNINGS

SURVANTA is intended for intratracheal use only.

SURVANTA CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE. Therefore, its use should be restricted to a highly supervised clinical setting with immediate availability of clinicians experienced with intubation, ventilator management, and general care of premature infants. Infants receiving SURVANTA should be frequently monitored with arterial or transcutaneous measurement of systemic oxygen and carbon dioxide.

DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED. If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure.

PRECAUTIONS

General

Rales and moist breath sounds can occur transiently after administration. Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present.

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3). The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants. The causative organisms were similar in treated and control infants. There was no significant difference between groups in the rate of post-treatment infections other than sepsis.

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials. There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation).

No information is available on the effects of doses other than 100 mg phospholipids/kg, more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Reproduction studies in animals have not been completed. Mutagenicity studies were negative. Carcinogenicity studies have not been performed with SURVANTA.

ADVERSE REACTIONS

The most commonly reported adverse experiences were associated with the dosing procedure. In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11.9% of doses. Oxygen desaturation occurred with 9.8% of doses.

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea. No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment.

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials. The rates in all controlled studies are in Table 3.

TABLE 3

	All Controlled Studies		
Concurrent Event	**SURVANTA (%)**	**Control (%)**	***P*-Value[a]**
Patent ductus arteriosus	46.9	47.1	0.814
Intracranial hemorrhage	48.1	45.2	0.241
Severe intracranial hemorrhage	24.1	23.3	0.693
Pulmonary air leaks	10.9	24.7	<0.001
Pulmonary interstitial emphysema	20.2	38.4	<0.001
Necrotizing enterocolitis	6.1	5.3	0.427
Apnea	65.4	59.6	0.283
Severe apnea	46.1	42.5	0.114
Post-treatment sepsis	20.7	16.1	0.019
Post-treatment infection	10.2	9.1	0.345
Pulmonary hemorrhage	7.2	5.3	0.166

[a]*P*-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage. However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63.3% *v* 30.8%, $P = 0.001$; and 48.8% *v* 34.2%, $P = 0.047$, respectively). The rate in a Treatment IND involving approximately 4400 infants was lower than in the controlled trials.

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests: white blood cell count and serum sodium, potassium, bilirubin, creatinine.

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C. No IgG or IgM antibodies were detected.

Several other complications are known to occur in premature infants. The following conditions were reported in the controlled clinical studies. The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA.

Respiratory: lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure.

Cardiovascular: hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardiorespiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return.

Gastrointestinal: abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer.

Renal: renal failure, hematuria.

Hematologic: coagulopathy, thrombocytopenia, disseminated intravascular coagulation.

Central Nervous System: seizures.

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia.

Musculoskeletal: inguinal hernia.

Systemic: fever, deterioration.

Follow-Up Evaluations

To date, no long-term complications or sequelae of SURVANTA therapy have been found.

Single-Dose Studies

Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations.

Multiple-Dose Studies

Six-month adjusted age follow-up evaluations have not been completed. Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants. Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy.

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants. To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results.

OVERDOSAGE

Overdosage with SURVANTA has not been reported. Based on animal data, overdosage might result in acute airway obstruction. Treatment should be symptomatic and supportive.

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage. Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present.

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venous blood. We investigated whether inhaling nitric oxide gas would cause selective vasodilation of ventilated lung regions, thereby reducing pulmonary hypertension and improving gas exchange. METHODS: Nine of 10 consecutive patients with severe ARDS inhaled nitric oxide in two concentrations for 40 minutes each. Hemodynamic variables, gas exchange, and ventilation-perfusion distributions were measured by means of multiple inert-gas-elimination techniques during nitric oxide inhalation; the results were compared with those obtained during intravenous infusion of prostacyclin. Seven patients were treated with continuous inhalation of nitric oxide in a concentration of 5 to 20 parts per million (ppm) for 3 to 53 days. RESULTS: Inhalation of nitric oxide in a concentration of 18 ppm reduced the mean (±SE) pulmonary-artery pressure from 37±3 torr to 30±2 torr ($p = 0.008$) and decreased intrapulmonary shunting from 36± 5% to 31±5% ($p = 0.028$). The ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen (P_{aO_2}/F_{IO_2}), an index of the efficiency of arterial oxygenation, increased during nitric oxide administration from 152±15 torr to 199±23 torr ($p = 0.008$), although the mean arterial pressure and cardiac output were unchanged. Infusion of prostacyclin reduced pulmonary-artery pressure but increased intrapulmonary shunting and reduced the P_{aO_2}/F_{IO_2} and systemic arterial pressure. Continuous nitric oxide inhalation consistently lowered the pulmonary-artery pressure and augmented the P_{aO_2}/F_{IO_2} for 3 to 53 days. CONCLUSIONS: Inhalation of nitric oxide by patients with severe ARDS reduces the pulmonary-artery pressure and increases arterial oxygenation by improving the matching of ventilation with perfusion, without producing systemic vasodilation. Randomized, blinded trials will be required to determine whether inhaled nitric oxide will improve outcome.

Respiratory Mechanics and Bronchodilator Responsiveness in Patients with the Adult Respiratory Distress Syndrome—A Pesenti, P Pelosi, N Rossi, M Aprigliano, L Brazzi, R Fumagalli. Crit Care Med 1993;21:78.

OBJECTIVE: To study the effects of salbutamol (a selective β_2-adrenergic receptor agonist) on respiratory mechanics in patients with the adult respiratory distress syndrome (ARDS). DESIGN: Prospective study. SETTING: ICU in a university hospital. PATIENTS: Seven mechanically ventilated, paralyzed ARDS patients. MAIN OUTCOME MEASUREMENTS: Measurements of respiratory system compliance, maximum and minimum inspiratory resistance (by the end-inspiratory occlusion method during constant flow inflation) were performed at 0, 5, 10 cm H_2O positive end-expiratory pressure, both before and at least 30 mins after the start of a continuous I.V. infusion of salbutamol (15 μg/min). Minimum inspiratory resistance represents the ohmic airflow resistance, while maximum inspiratory resistance includes minimum inspiratory resistance plus the effective additional resistance due to stress adaptation and to time constant inhomogeneities. Airflow was measured at the airway connector and tracheal pressure near the central end of the artificial airway. RESULTS: Maximum inspiratory resistance, minimum inspiratory resistance, and additional resistance were higher than the values reported for normal anesthetized subjects. On average, salbutamol caused a decrease in maximum and minimum inspiratory resistances (from 6.48 ± 2.56 to 4.67 ± 1.74 and from 4.06 ± 2.12 to 2.07 ± 0.95 cm $H_2O \cdot s \cdot L^{-1}$, respectively). Positive end-expiratory pressure increased additional resistance, whereas it decreased minimum inspiratory resistance. No interaction was found between positive end-expiratory pressure and salbutamol. Respiratory system compliance was not significantly affected by salbutamol nor by positive end-expiratory pressure. CONCLUSIONS: In ARDS patients, salbutamol decreases the abnormally high airway resistance, by reducing minimum resistance, but has no effect on the effective additional resistance.

Genetic Determinants of Airways' Colonisation with *Pseudomonas aeruginosa* in Cystic Fibrosis—P Kubesch, T Dörk, U Wulbrand, N Kälin, T Neumann, B Wulf, et al. Lancet 1993;341:189.

Exocrine pancreatic insufficiency and lung infection with *Pseudomonas aeruginosa* are major features of cystic fibrosis (CF). This monogenic disease is caused by mutations in the CF transmembrane conductance regulator (CFTR) gene. 267 children and adolescents with CF who were regularly seen at the same centre were assessed for an association of the CFTR mutation genotype with exocrine pancreatic function and the age of onset of chronic colonisation with *P aeruginosa*. The major mutation ΔF508 accounted for 74% of CF alleles; 33 further CFTR mutations had been detected on the CF chromosomes of the study population by June, 1992. With the exception of ΔF508/R347P compound heterozygotes, patients of the same mutation genotype were either pancreas insufficient (PI) or pancreas sufficient (PS). The age-specific colonisation rates with *P aeruginosa* were significantly lower in PS than in PI patients. The missense and splice site mutations that are "mild" CF alleles with respect to exocrine pancreatic function were also "low risk" alleles for the acquisition of *P aeruginosa*. On the other hand, the proportion of *P aeruginosa*-positive

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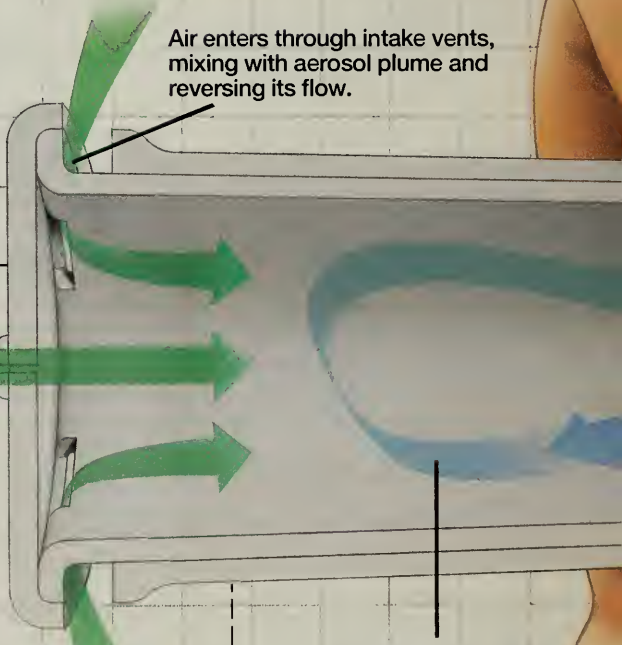
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patients increased most rapidly in the PI ΔF508 compound heterozygotes who were carrying a termination mutation in the nucleotide binding fold-encoding exons. Pancreatic status and the risk of chronic airways' colonisation with *P aeruginosa* are predisposed by the CFTR mutation genotype and can be differentiated by the type and location of the mutations in the CFTR gene.

Iatrogenic Complications in Adult Intensive Care Units: A Prospective Two-Center Study—T Giraud, J-F Dhainaut, J-F Vaxelaire, T Joseph, D Journois, G Bleichner, et al. Crit Care Med 1993;21:40.

OBJECTIVES: (a) To evaluate the frequency, types, severity, and morbidity of iatrogenic complications; (b) determine associated factors that favor iatrogenic complications; and (c) suggest new or more efficient protective measures that may be taken to improve patient safety. DESIGN: Prospective, observational study. SETTING: Two ICUs in France. PATIENTS & METHODS: The study included 382 patients (age ≥ 15 yrs; 400 consecutive admissions). Patients were monitored by two physicians in each ICU to assess all iatrogenic complications occurring during their ICU stay, with the exception of adverse effects of drugs. An iatrogenic complication was defined as an adverse event that was independent of the patient's underlying disease. RESULTS: We observed 316 iatrogenic complications in 124 (31%) of the 400 admissions. Of these iatrogenic complications, 107 (in 53 [13%] of the 400 admissions) complications were major, three leading to death. Severe hypotension, cardiac arrest represented 78% of the major iatrogenic complications. Fifty-nine per cent of the major iatrogenic complications had clearly identified associated factors. Human errors accounted for 67% of these factors. Patients > 65 yrs (adjusted odds ratio = 2.6, 95% confidence interval: 1.4 to 4.9) and those patients admitted with two or more organ failures (adjusted odds ratio = 4.8, 95% confidence interval: 2.5 to 9.2) were more likely to develop major iatrogenic complications. High or excessive nursing workload also led to an increased risk of major iatrogenic complications. Persistent morbidity, secondary to iatrogenic complications at the time of discharge, was present in 5 survivors. The risk of ICU death was about 2-fold higher for the patients with major iatrogenic complications than in the remaining patients after adjusting for the Organ System Failure Score and the prognosis of the disease (relative risk = 1.92, 95% confidence interval: 1.28 to 2.56). CONCLUSIONS: Major iatrogenic complications were frequent, associated with increased morbidity and mortality rates, related to high or excessive nursing workload, and were often secondary to human errors. To improve patient safety in our ICUs, preventive measures should be targeted primarily on the elderly and the most severely ill patients. Special attention should be given to improving the organization of workload and training, and promoting wider use of noninvasive monitoring.

Predicting Mortality Risk for Infants Weighing 501 to 1,500 Grams at Birth: A National Institutes of Health Neonatal Research Network Report—JD Horbar, L Onstad, E Wright, and The National Institute of Child Health and Human Development Neonatal Research Network. Crit Care Med 1993;21:12.

OBJECTIVES: To develop and evaluate a model that predicts mortality risk based on admission data for infants weighing 501 to 1,500 grams at birth, and to use the model to identify neonatal ICUs where the observed mortality rate differs significantly from the predicted rate. DESIGN: Validation cohort study. SETTING: University-based, tertiary care neonatal ICUs. PATIENTS: Sample of 3,603 infants with birthweights of 501 to 1,500 grams who were born at seven National Institute of Child Health and Human Development (NICHHD) Neonatal Research Network Centers, over a 2-yr period of time. INTERVENTIONS: None. MEASUREMENTS & MAIN RESULTS: Based on logistic regression analysis, admission factors associated with mortality risk for inborn infants were: decreasing birthweight, appropriate size for gestational age, male gender, non-black race, and 1-min Apgar score of ≤ 3. The mortality prediction model based on these factors had a sensitivity of 0.50, a specificity of 0.92, a correct classification rate of 0.82, and an area under the receiver operating characteristic curve of 0.82 when applied to a validation sample. Goodness-of-fit testing showed that there was a marginal degree of fit between the observations and model predictions ($\chi^2 = 15.4$, $p = 0.06$). The observed mortality rate for 3,603 infants at the seven centers was 24.7%, ranging from 21.8% to 27.7% at individual centers. There were no statistically significant differences between observed and predicted mortality rates at any of the centers. One center had an observed mortality rate that was 2.8% lower than predicted by the model (95% confidence interval –6.0% to 0.5%), and another center had an observed rate that was 3% higher than expected (95% confidence interval –0.3% to 6.2%). CONCLUSIONS: Mortality risk for infants weighing 501 to 1500 grams can be predicted based on admission factors. However, until more accurate predictive models are developed and validated and the relationships between care practices and outcomes are better understood, such models should not be relied on for evaluating the quality of care provided in different neonatal ICUs.

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Mechanisms of Ventilator-Induced Lung Injury—JC Parker, LA Hernandez, KJ Peevy. Crit Care Med 1993;21:131.

OBJECTIVES: To describe the physiologic mechanisms of ventilator-induced lung injury and to define the major ventilator and host-dependent risk factors that contribute to such injury. DATA SOURCES: Basic science and clinical studies related to ventilator-induced barotrauma and lung pathophysiology. STUDY SELECTION: Emphasis on controlled, experimental studies and clinical studies related to specific mechanisms. DATA EXTRACTION: Preference given to studies with quantitative end points to assess damage and causal relationships. DATA SYNTHESIS: Related studies are integrated to obtain basic mechanisms of damage where possible. CONCLUSIONS: Ventilation with high tidal volumes can increase vascular filtration pressures; produce stress fractures of capillary endothelium, epithelium, and basement membrane; and cause lung rupture. Mechanical damage leads to leakage of fluid, protein, and blood into tissue and air spaces or leakage of air into tissue spaces. This process is followed by an inflammatory response and possibly a reduced defense against infection. Predisposing factors for lung injury are high peak inspiratory volumes and pressures, a high mean airway pressure, structural immaturity of lung and chest wall, surfactant insufficiency or inactivation, and preexisting lung disease. Damage can be minimized by preventing overdistention of functional lung units during therapeutic ventilation.

Effects of Dense, High-Volume, Artificial Surfactant Aerosol on a Heated Exhalation Filter System—CF Haas, JG Weg, CW Kettell, EJ Caldwell, DS Zaccardelli, DL Brown. Crit Care Med 1993;21:125.

OBJECTIVE: To evaluate a supplemental heated filter system during mechanical ventilation with continuous nebulization of an artificial surfactant by a new, high-volume nebulizer. DESIGN & METHODS: A new nebulizer system, containing artificial surfactant, provided half of a 20-L minute ventilation and the remainder of this minute ventilation was provided by a commonly used mechanical ventilator. Ventilation sources were joined in the inspiratory limb of the breathing circuit, which was connected to a test lung system. A supplemental filter system was placed upstream of the ventilator's heated filter in the expiratory limb of the circuit. Circuit pressures at the inlet of the supplemental filter (P1), between the filters (P2), and after the ventilator expiratory filter (P3) were monitored and recorded. Nebulizer canisters containing artificial surfactant were replaced every 4 hrs. The performance of four supplemental filters in continuous use was examined. Another four filters were each used over 4 hrs, steam autoclaved, and reused. SETTINGS: The ventilator was set at a rate of 20 breaths/min, with a tidal volume of 0.5 L, a flowrate of 40 L/min, and positive end-expiratory pressure of 10 cm H_2O. The nebulizer provided an equal volume and flowrate so that the delivered tidal volume was 1.0 L with a flowrate of 80 L/min. RESULTS: Ventilator failure and/or excessive airway pressure caused by increased filter resistance occurred at a mean of 7.3 ± 1.3 (SD) hrs of continuous ventilation. Mean P1-peak increased from 67.5 ± 8.2 to 94.0 ± 10.7 cm H_2O ($p < 0.001$) and P1-baseline increased from 9.3 ± 1.0 to 53.5 ± 17.1 cm H_2O ($p = 0.014$). Filters that were autoclaved after 4 hrs of ventilation and reused lasted a total of 7.0 ± 1.3 hrs. Mean P1-peak increased from 68.9 ± 4.9 to 84.8 ± 19.1 cm H_2O and P1-baseline increased from 9.5 ± 1.7 to 30.8 ± 14.2 cm H_2O ($p < 0.05$). CONCLUSIONS: The supplemental filter system was able to protect the ventilatory exhalation sensors for approximately 7 hrs at a minute ventilation of 20 L/min. Steam sterilization did not extend the supplemental filter life.

Synchronous Mechanical Ventilation of the Neonate with Respiratory Disease—M Amitay, PC Etches, NN Finer, JM Maidens. Crit Care Med 1993;21:118.

OBJECTIVES: To assess the importance of synchronization of mechanical ventilation with spontaneous respiratory efforts in mechanically ventilated neonates. The actions of this synchronization on ventilation, oxygenation, and BP variation were assessed. DESIGN: Prospective evaluation using within-subject comparison of asynchronous and synchronous states. SETTING: Neonatal ICU in a large, university-affiliated hospital. PATIENTS: Fourteen neonates requiring mechanical ventilation who were initially asynchronous with the ventilator. INTERVENTION: The ventilator settings were adjusted using the patients' own inspiratory and expiratory timing to create synchronous interaction with the ventilator. MEASUREMENTS & MAIN RESULTS: Synchrony was assessed using clinical observation combined with inspection of the air flow waveform and computerized analysis of the air flow signal to assess cycle-to-cycle reproducibility, so-called autocorrelation. Synchronous ventilation significantly improved tidal volume ($p < 0.05$), minute volume ($p < 0.001$), and all indices of the variability of arterial BP ($p < 0.001$). Mean airway pressure did not change significantly. No infant developed an airleak syndrome or intraventricular hemorrhage, which have previously been associated with asynchronous ventilation and an unstable BP, re-

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spectively. CONCLUSION: Synchronous ventilation can be readily applied to most ventilated neonates. It improves ventilation, and results in a marked reduction in BP variation, which may have implications for reducing the risk of intraventricular hemorrhage.

Spirometry Reference Values for Women and Men 65 to 85 Years of Age: Cardiovascular Health Study—PL Enright, RA Kronmal, M Higgins, M Schenker, EF Haponik. Am Rev Respir Dis 1993;147:125.

Pulmonary function was assessed by spirometry in 5,201 ambulatory elderly participants of the Cardiovascular Health Study, sampled from four communities. A stringent quality assurance program exceeded American Thoracic Society (ATS) recommendations for spirometry. Less than 6% of the participants were unable to perform three acceptable spirometry maneuvers. A "healthy" subgroup of 777 women and men 65 to 85 yr of age was identified by excluding smokers and those with lung disease and other factors determined to independently, significantly, and negatively influence the FEV_1. Results from black participants were examined separately. Reference equations and normal ranges for FEV_1, FVC, and the FEV_1/FVC ratio were determined from the healthy group. The results demonstrate differences in predicted values as great as 20% (0.5 to 1 L) for elderly patients when compared with the spirometry reference equations that are most commonly used in the United States.

Hypoplastic Trachea in Down's Syndrome—LS Aboussouan, PB O'Donovan, DS Moodie, LA Gragg, JK Stoller. Am Rev Respir Dis 1993; 147:72.

Our study sought to determine whether tracheal dimensions were reduced in patients with Down's syndrome and whether such a narrowing, if present, was related to the presence of congenital heart disease or to body habitus (height or weight). The inner diameter of the tracheal air column was measured at 2 cm above the aortic arch in 14 adult patients with Down's syndrome, and the results compared with previously established norms. Two-way analysis of variance showed no significant difference in z-scores between sexes or between those with or without congenital heart disease. Z-scores were significantly different from zero for both coronal diameters ($p = 0.0010$) and sagittal diameters ($p = 0.0003$). The negative limits on the 95% confidence interval for coronal z-scores (–2.5 to –0.8) and sagittal z-scores (–2.6 to –1.0) indicate that our patients have tracheal diameters significantly smaller than normal. Linear regression analysis showed no significant correlation between tracheal diameters and patients' height or weight. We conclude that tracheal diameters in adult patients with Down's syndrome are reduced and that the narrowing cannot be ascribed to associated congenital heart disease or to body habitus.

A Comparison of Clinical Assessment and Home Oximetry in the Diagnosis of Obstructive Sleep Apnea—S Gyulay, LG Olson, MJ Hensley, MT King, KM Allen, NA Saunders. Am Rev Respir Dis 1993;147: 50.

In order to determine whether measurement of arterial oxygen saturation (S_{aO_2}) could identify patients with obstructive sleep apnea (OSA), 98 consecutive patients referred for assessment of snoring and/or daytime somnolence were assessed clinically and then underwent both unsupervised oximetry in their homes and formal polysomnography. Clinical assessment identified patients with an apnea + hypopnea index (AHI) $\geq$ 15 events per hour with a sensitivity of 79% and a specificity of 50%. Home oximetry analyzed by counting the number of arterial oxygen desaturations recorded was inferior to clinical assessment. For desaturations of 2% or more from baseline, desaturation index (DI) $\geq$ 15 per hour identified patients with AHI $\geq$ 15 with sensitivity 65% and specificity 74%; for 3% desaturations, sensitivity was 51% and specificity 90%; and for 4% desaturations, sensitivity was 40% and specificity 98%. From the oximetry data, the percentage of time spent at S_{aO_2} below 90% (CT_{90}) was also calculated. $CT_{90} \geq 1\%$ identified patients with AHI $\geq$ 15 with sensitivity 93% and specificity 51%; for patients with AHI $\geq$ 15 ultimately given nasal continuous positive airway pressure (CPAP), the sensitivity of a $CT_{90} \geq 1\%$ was 100%. We concluded that home oximetry with $CT_{90} < 1\%$ practically excludes clinically significant OSA. Conversely, home oximetry with DI $\geq$ 15 for 4% desaturations makes OSA likely: The positive predictive value for OSA is 83% if the pretest probability of OSA is 30% and over 90% if the pretest probability is at least 50%.

Physiologic Effects of Positive End-Expiratory Pressure in Patients with Chronic Obstructive Pulmonary Disease during Acute Ventilatory Failure and Controlled Mechanical Ventilation—VM Ranieri, R Giuliani, G Cinnella, C Pesce, N Brienza, EL Ippolito, et al. Am Rev Respir Dis 1993;147:5.

Dynamic hyperinflation and intrinsic positive end-expiratory pressure ($PEEP_i$) are observed in patients with chronic obstructive pulmonary disease (COPD) and flow limitation. Several reports suggest that PEEP levels approaching $PEEP_i$ reduce inspiratory load due to $PEEP_i$, without further hyperinflation. Hence, PEEP

should not increase intrathoracic pressure or affect hemodynamics and gas exchange. To verify this hypothesis, the effects of PEEP (0 to 15 cm H_2O) on respiratory mechanics, hemodynamics, and gas exchange were studied in 9 COPD patients during controlled mechanical ventilation. PEEP levels approaching $PEEP_i$ (9.8 ± 0.5 cm H_2O) did not affect the expiratory flow/volume relationship, confirming the presence of flow limitation. PEEP levels of 5 and 10 cm H_2O did not change lung volume and $PEEP_i$ in the respiratory system ($PEEP_{tot,rs}$) and chest wall ($PEEP_{tot,cw}$) or affect hemodynamics and gas exchange. When applied PEEP overcame $PEEP_i$, changes in lung volume and the expiratory flow/volume relationship were observed. $PEEP_{tot,rs}$ and $PEEP_{tot,cw}$ also increased. Under these circumstances, PEEP increased static elastance in both the respiratory system and the chest wall, reducing cardiac index and affecting hemodynamics and gas exchange. Our data show that in mechanically ventilated COPD patients with $PEEP_i$ due to flow limitation, PEEP levels exceeding the 85% of $PEEP_i$ (P_{crit}) caused further hyperinflation and compromised hemodynamics and gas exchange.

A General Mathematical Model for Respiratory Dynamics Relevant to the Clinical Setting—JJ Marini, PS Crooke. Am Rev Respir Dis 1993;147:14.

We have developed and validated a general mathematical model for the dynamic behavior of the single-compartment respiratory system in response to an arbitrary waveform of applied inspiratory pressure. Our general model for ventilation applies to all integrable functions of applied pressure, and it enables computation of most ventilation and pressure variables of clinical interest from clinician-selected and impedance de-

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termined inputs readily measured or estimated at the bedside. Interactions between both phases of the ventilatory cycle are considered by assuming that deflation occurs passively from a unicompartment lung. Because this flexible model appears both capable of accurate prediction and robust to major violations of its underlying linear assumptions, it may prove to be of value in a variety of scientific, educational, and clinical settings.

Oxygen Consumption Is Independent of Increases in Oxygen Delivery by Dobutamine in Septic Patients Who Have Normal or Increased Plasma Lactate—JJ Ronco, JC Fenwick, BR Wiggs, PT Phang, JA Russell, MG Tweeddale. Am Rev Respir Dis 1993;147:25.

We asked whether the relationship between oxygen delivery and oxygen consumption is different between patients who have sepsis and normal (n = 6) or increased (n = 8) concentrations of plasma lactate. We determined oxygen consumption using analysis of respiratory gases while increasing oxygen delivery using a dobutamine infusion. The relationship between oxygen delivery and consumption was $y = 124 + 0.043 * x$ in the normal lactate group and $y = 131 - 0.003 * x$ in the high lactate group (95% CI for differences in slopes, –0.003 to 0.096; $p \leq 0.05$ for slope, normal versus high lactate). In the normal lactate group, direct oxygen consumption increased by only 8 ± 6 mL · min · m^{-2} after dobutamine infusion (from 144 ± 26 to 153 ± 22 mL · min · m^{-2}, $p \leq 0.02$) despite an average increase of 220 ± 80 mL · min · m^{-2} in oxygen delivery (from 446 ± 91 to 666 ± 90 mL · min · m^{-2}, $p \leq 0.01$). The oxygen extraction ratio fell from 0.27 ± 0.03 to 0.21 ± 0.02 after dobutamine ($p \leq 0.017$). In the high lactate group, direct oxygen consumption decreased by 1 ± 6 mL · min · m^{-2} after dobutamine (from 131 ± 33 to 130 ± 35 mL · min · m^{-2}, $p > 0.60$) despite an average increase of 168 ± 138 mL · min · m^{-2} in oxygen delivery (from 467 ± 194 to 635 ± 300 mL · min · m^{-2}, $p \leq 0.01$). The oxygen extraction ratio fell from 0.30 ± 0.14 to 0.26 ± 0.12 after dobutamine ($p \leq 0.01$). We conclude first that direct oxygen consumption remains relatively constant despite large increases in oxygen delivery in patients who have sepsis, and second that increased concentration of plasma lactate does not predict dependence of oxygen consumption on oxygen delivery.

Meta-Analysis of the Literature or of Individual Patient Data: Is There a Difference?—LA Stewart, MKB Parmar. Lancet 1993;341:418.

The use of meta-analyses or overviews to combine formally the results of related randomised clinical trials is becoming increasingly common. However, the distinction between analyses based on information extracted from the published literature and those based on collecting and reanalysing updated individual patient data is not clear. We have investigated the difference between meta-analysis of the literature (MAL) and meta-analysis of individual patient data (MAP) by comparing the two approaches using randomised trials of cisplatin-based therapy in ovarian cancer. The MAL was based on 788 patients and the MAP on 1,329 and estimated median follow-ups were 3.5 and 6.5 years, respectively. The MAL gave a result of greater statistical significance ($p = 0.027$ vs $p = 0.30$) and an estimate of absolute treatment effect three times as large as the MAP (7.5% vs 2.5%). Publication bias, patient exclusion, length of follow-up, and method of analysis all contributed to this observed difference. The results of a meta-analysis of the literature alone may be misleading. Whenever possible, a meta-analysis of updated individual patient data should be done because this provides the least biased and most reliable means of addressing questions that have not been satisfactorily resolved by individual clinical trials.

Tolerance to the Nonbronchodilator Effects of Inhaled β_2-Agonists in Asthma—BJ O'Connor, SL Aikman, PJ Barnes. N Engl J Med 1992; 327:1204.

BACKGROUND: Tolerance to the direct bronchodilator effects of β_2-agonists does not appear to occur in asthma. However, it is not known whether this is true for the nonbronchodilator effects of these agents, which protect the airways against bronchoconstrictive stimuli. METHODS: We investigated whether tolerance develops to the protective effect of inhaled terbutaline on airway responsiveness to the bronchoconstrictors methacholine (which acts directly on airway smooth muscle) and AMP (which acts indirectly by stimulating the release of mediators from mast cells) during sustained treatment with terbutaline. In a randomized, double-blind, crossover study, 12 patients with mild asthma each inhaled a single dose of terbutaline (500 μg) or placebo before a challenge with a series of doubling doses of inhaled methacholine or AMP, before and after treatment for 7 days with 500 μg of terbutaline four times daily or placebo. RESULTS: Before the 7 days of treatment with terbutaline, a single dose of terbutaline reduced airway responsiveness to methacholine by 2.7 doubling doses (95% confidence interval, 1.9-3.5), but it had an even greater protective effect against AMP, reducing airway

responsiveness by 3.8 doubling doses (95% confidence interval, 2.7-4.9, $p < 0.001$). After 7 days of treatment with terbutaline, the protective effect of terbutaline against methacholine decreased to 2.2 doubling doses (95% confidence interval, 1.3-3.0 $p = 0.04$), and that against AMP decreased even more to 1.7 doubling doses (95% confidence interval 1.1-2.4; $p < 0.001$). By contrast, the bronchodilator response to terbutaline was unchanged during 7 days of treatment with this agent. CONCLUSIONS: We observed tolerance to the nonbronchodilator actions of the inhaled β_2-agonist terbutaline in patients with mild asthma, an effect that may be more pronounced in mast cells than in bronchial smooth muscle. This property of β_2-agonists may constitute a drawback to their regular use in patients with asthma.

Long-Term Effects of a Long-acting β_2-Adrenoceptor Agonist, Salmeterol, on Airway Hyperresponsiveness in Patients with Mild Asthma—D Cheung, MC Timmers, AH Zwinderman, EH Bel, JH Dijkman, PJ Sterk. N Engl J Med 1992; 327:1198.

BACKGROUND: Asthma is characterized by hyperresponsiveness of the airways to bronchoconstrictive stimuli. Long-acting β_2-adrenoceptor agonists have been introduced as a new therapeutic approach, but there is growing concern about whether control of asthma may deteriorate with the regular use of these agents. We investigated the long-term effects of the β_2-agonist salmeterol on bronchodilation and on airway hyperresponsiveness to the bronchoconstrictive agent methacholine in mild asthma. METHODS: In a parallel, double-blind study, 24 patients with mild asthma were randomly assigned to treatment with either inhaled salmeterol (50 µg, twice daily) (n = 12) or placebo (n = 12) during an 8-week trial. Methacholine challenge was performed before, during, and after the treatment period. Methacholine responsiveness was measured as the provocative concentration (PC_{20}) that caused a 20% decrease in the forced expiratory volume in one second (FEV_1). RESULTS: There was a significant increase in FEV_1 one hour after the inhalation of salmeterol ($p = 0.006$), which did not differ significantly on Days 0, 28, and 56 of the treatment period (increase, 9.8, 9.4, and 8.8% of predicted FEV_1, respectively; $p = 0.91$). On the first treatment day, salmeterol afforded significant protection against metha-

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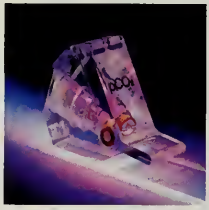

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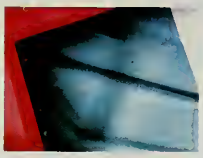

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choline-induced bronchoconstriction, as shown by a 10-fold increase in the PC_{20} as compared with the value at entry ($p < 0.001$). After 4 and 8 weeks of treatment, however, the salmeterol-induced change in the PC_{20} was significantly attenuated ($p < 0.001$) to only a two-fold increase. Two and four days after treatment ended, the PC_{20} was not significantly different from the value before treatment ($p = 0.15$). CONCLUSIONS: Regular treatment of patients with mild asthma with salmeterol leads to tolerance to its protective effects against a bronchoconstrictor stimulus, in this case inhaled methacholine, despite well-maintained bronchodilation. This finding raises concern about the effectiveness of prolonged therapy with long-acting β_2-adrenoceptor agonists in asthma.

Long-Term Effects of a Long-Acting β_2-Adrenoceptor Agonist, Salmeterol, on Airway Hyperresponsiveness in Patients with Mild Asthma—D Cheung, MC Timmers, AH Zwinderman, EH Bel, JH Dijkman, PJ Sterk. N Engl J Med 1992; 327:1198.

BACKGROUND: Asthma is characterized by hyperresponsiveness of the airways to bronchoconstrictive stimuli. Long-acting β_2-adrenoceptor agonists have been introduced as a new therapeutic approach, but there is growing concern about whether control of asthma may deteriorate with the regular use of these agents. We investigated the long-term effects of the β_2-agonist salmeterol on bronchodilation and on airway hyperresponsiveness to the bronchoconstrictive agent methacholine in mild asthma. METHODS: In a parallel, double-blind study, 24 patients with mild asthma were randomly assigned to treatment with either inhaled salmeterol (50 μg, twice daily) ($n = 12$) or placebo ($n = 12$) during an 8-week trial. Methacholine challenge was performed before, during, and after the treatment period. Methacholine responsiveness was measured as the provocative concentration (PC_{20}) that caused a 20% decrease in the forced expiratory volume in 1 second (FEV_1). RESULTS: There was a significant increase in FEV_1 1 hour after the inhalation of salmeterol ($p = 0.006$), which did not differ significantly on Days 0, 28, and 56 of the treatment period (increase, 9.8, 9.4, and 8.8% of predicted FEV_1, respectively; $p = 0.91$). On the first treatment day, salmeterol afforded significant protection against methacholine-induced bronchoconstriction, as shown by a 10-fold increase in the PC_{20} as compared with the value at entry ($p < 0.001$). After 4 and 8 weeks of treatment, however, the salmeterol-induced change in the PC_{20} was significantly attenuated ($p < 0.001$) to only a two-fold increase. Two and four days after treatment ended, the PC_{20} was not significantly different from the value before treatment ($p = 0.15$). CONCLUSIONS: Regular treatment of patients with mild asthma with salmeterol leads to tolerance to its protective effects against a bronchoconstrictor stimulus, in this case inhaled methacholine, despite well-maintained bronchodilation. This finding raises concern about the effectiveness of prolonged therapy with long-acting β_2-adrenoceptor agonists in asthma.

The Effect of Age on Bronchodilator Responsiveness—DJ Turner, LI Landau, PN LeSouëf. Pediatr Pulmonol 1993;15:98.

The relationship between age and bronchodilator responsiveness (BDR) in children has not been studied using objective parameters. The aim of this study was to seek such a relationship in young asthmatic children using dose-response curves (DRC). Fourteen asthmatic subjects (age 3-9 years) with a forced expiratory volume in 1 sec (FEV_1) less than 80% predicted were studied after being trained to use a spirometer reliably. Each subject completed a DRC by inhaling 5 doses of salbutamol (albuterol) at 15 min intervals until a cumulative total of 6.84 mg of salbutamol had been administered. FEV_1, forced vital capacity (FVC), and forced expiratory flow at mid vital capacity (FEF_{25-75}) were measured before and after each nebulization. In addition, arterial oxygen saturation (S_{aO_2}) and heart rate (HR) were measured in some of the subjects. All lung function parameters, S_{aO_2}, and HR increased significantly between baseline and completion of the DRC. A significant age effect on BDR was detected in FEV_1 and FVC, with older children showing a greater response than young ones. The response had plateaued after the maximum dose in the younger but not in the older children. These findings suggest that the level of response to a bronchodilator increases significantly with increasing age in young asthmatics.

Pulmonary Function between 6 and 18 Years of Age—X Wang, DW Dockery, D Wypij, ME Fay, BG Ferris Jr. Pediatr Pulmonol 1993; 15:75.

Pulmonary function of children aged 6-18 years is described based on 82,462 annual measurements of forced vital capacity (FVC), forced expired volume in 1 second (FEV_1), and forced expiratory flow between 25% and 75% of FVC ($FEF_{25-75\%}$) from 11,630 White children and 989 Black children. Median height, FVC, FEV_1, FEV_1/FVC, and $FEF_{25-75\%}$ for each 3 months of age are compared among race and sex subgroups. Race- and sex-specific percentile distributions of FVC, FEV_1, FEV_1/FVC, and $FEF_{25-75\%}$ are presented for each centimeter of height (growth curves). For the same height, boys have great-

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er lung function values than girls, and Whites have greater ones than Blacks. Lung function increases linearly with age until the adolescent growth spurt at about age 10 years in girls and 12 in boys. The pulmonary function vs height relationship shifts with age during adolescence. Thus, a single equation or the pulmonary function-height growth chart alone does not completely describe growth during the complex adolescent period. Nevertheless, race- and sex-specific growth curves of pulmonary function vs height make it easy to display and evaluate repeated measures of pulmonary function for an individual child. Race-, sex-, and age-specific regression equations based on height are provided, which permit the evaluation of growth during adolescence with improved accuracy and, more importantly, in comparison with previous observations for the same child.

Pulmonary Mechanics and Gas Exchange: Effect of Lateral Positioning during Recovery from Respiratory Distress Syndrome—JS Schlessel, HA Rappa, M Lesser, RG Harper. Pediatr Pulmonol 1993;15: 36.

Sixteen stable intubated premature infants without a clinically significant patent ductus arteriosus were studied during recovery from respiratory distress syndrome in order to determine the effects of left and right lateral, as compared to supine, positioning. Pulmonary mechanics were measured for spontaneous breaths 5 and 15 minutes after positioning, and arterial blood gases 15 minutes after positioning. Infants were randomized to 1 of 2 position sequences: (1) supine, left, supine, right or (2) supine, right, supine, left. No significant differences were detected between positions for dynamic compliance, tidal volume/kg, and total, inspiratory and expiratory pulmonary resistance. Likewise, no significant differences in P_{aO_2} or P_{aCO_2} were detected between the positions. The sequence of positions did not affect the pulmonary mechanics of spontaneous breaths or arterial blood gases. This suggests that short-term lateral positioning as well as supine positioning can be utilized without deleterious effects on pulmonary mechanics and gas exchange in neonates recovering from respiratory distress syndrome.

Hypoxemia during Altitude Exposure: A Meta-Analysis of Chronic Obstructive Pulmonary Disease—TA Dillard, AP Rosenberg, BW Berg. Chest 1993;103:422.

A previous study identified spirometric testing as a useful adjunct for estimating P_{aO_2} during altitude exposure in patients with chronic obstructive pulmonary disease (COPD). We sought to examine the validity of this finding by quantitative analysis of recent published reports. We analyzed acute hypoxic exposures from five prior studies involving 71 patients. Across all studies, the change in arterial oxygen tension per unit change in inspired oxygen partial pressure (linear slope, dP_{aO_2}/dP_{IO_2}) correlated with the pre-exposure forced expiratory volume in 1 s (FEV_1, $p < 0.01$). The correlation with FEV_1 held for values weighted or unweighted by sample size, with rotating deletion of each study from analysis one at a time, and with semilog slope as the dependent variable. A formula derived from the semilog slope relationship with FEV_1 gave accurate description of the mean hypoxic response in each prior study and individual responses from one study (n = 18):

$$\ln (P_{aO_2}alt/P_{aO_2}g) = k_n \cdot (P_{IO_2}alt - P_{IO_2}g).$$

We found that FEV_1 modulated the values of k_n in this study. We conclude, based on analysis of prior studies, that pre-exposure arterial oxygen tension and FEV_1 both influence the prediction of P_{aO_2} during hypoxic exposures in patients with COPD.

Assessment of Exercise Capacity in Asthmatic Children with Various Degrees of Activity—G Fink, C Kaye, H Blau, SA Spitzer. Pediatr Pulmonol 1993;15:41.

Physical fitness in a group of 49 stable asthmatic children was determined by an incremental exercise test. Thirty-one normal children served as a control group. The asthmatic children were divided into three groups. Group 1 was comprised of 16 children who actively participated in organized sports, Group 2 of 16 children who did not participate in organized sports but who engaged in free-play, and Group 3 of 17 children with a sedentary lifestyle who avoided even free-play. The results of cardiopulmonary evaluation before and after maximal incremental exercise testing have shown that Groups 1 and 2 behaved like the control group and their physical fitness was similar. Group 3 whose life-style was sedentary had poor physical fitness as compared to the other asthmatics and to the control group. This was the result of poor cardiovascular conditioning and was unrelated to the respiratory limitation. We conclude that poor physical fitness in asthmatic children is the result of a sedentary lifestyle and can be potentially normalized.

Rate of Decay or Increment of P_{aO_2} following a Change in Supplemental Oxygen in Mechanically Ventilated Patients with Diffuse Pneumonia—R Solis, C Anselmi, M Lavietes, MA Khan. Chest 1993;103: 554.

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It has been shown that patients with COPD require as long as 20 min for equilibration of oxygen tension to occur after changing the fraction of inspired oxygen (F_{IO_2}). To date, there have been no studies to determine the equilibration time for the P_{aO_2} in mechanically ventilated patients with diffuse pneumonia. We studied 7 patients (5 males, 2 females) with radiographic evidence of diffuse pneumonia. All patients required mechanical ventilation. After introducing a change in F_{IO_2}, arterial blood gas values were measured at 5-min intervals for 30 min. Four patients achieved maximal change in P_{aO_2} after 5 min, while four patients required 10 min. These results are similar to those found in patients with left ventricular failure who experience equilibration rapidly; however, patients with COPD experience it at a much slower pace. These observations have clinical importance when managing unstable patients where time is a critical element.

Mechanical Ventilation for the Elderly Patient in Intensive Care: Incremental Charges and Benefits—IL Cohen, J Lambrinos, IA Fein. JAMA 1993;269:1025.

OBJECTIVE: To evaluate the cost-effectiveness of prolonged mechanical ventilation in patients 80 years of age and older in the intensive care unit (ICU). DESIGN: A retrospective review of consecutive ICU patients requiring 3 or more days of mechanical ventilation. Cost-effectiveness analysis was performed by assessing incremental hospital charges from hospital billing records; charges were then related to years of life saved. A telephone survey was used to follow up hospital survivors for a minimum of 4 years after discharge. SETTING: A 20-bed medical-surgical ICU in a 420-bed, tertiary-care community teaching hospital. PATIENTS: The study included all patients aged 80 years or older taken from a comprehensive database of all patients admitted to the ICU requiring mechanical ventilation from April 1, 1985, through October 31, 1987 (n = 512). Of 59 potential candidates, 45 were found to have complete billing records and were the subject of further analysis. RESULTS: Of the 45 patients in the group under analysis, 10 survived to leave the hospital. Of these, two were alive and one could not be located at the time of follow-up. The charge per year of life saved is estimated to be between $51,854 and $75,090 in 1985-1987 dollars. Of 22 patients whose age in years plus duration of mechanical ventilation in days totaled 100 or greater, only two survived hospitalization and neither was alive at follow-up. The cost per year of life saved in this subset of patients was $181,308 in 1985-1987 dollars. One of these patients was discharged to a nursing home and died there 4.5 years later, after multiple hospital readmissions. The other patient died at home 2 months after hospital discharge. CONCLUSION: Based on hospital charges and life expectancy, the cost-effectiveness of prolonged mechanical ventilation in ICU patients age 80 years and over was poor in our population when the combination of age and duration of mechanical ventilation exceeded 100. Further studies using this type of analysis may prove valuable in both clinical and administrative decision-making processes.

Lung Recoil and the Determination of Airflow Limitation in Cystic Fibrosis and Asthma—A Zapletal, KJ Desmond, D Demizio, AL Coates. Pediatr Pulmonol 1993; 15:13.

A reduction of lung recoil pressure could aggravate any airflow limitation that might be present in patients with cystic fibrosis (CF) or asthma. In a group of 22 children and young adults (8 with cystic fibrosis, 8 with asymptomatic asthma, and 6 healthy controls) aged 8-24 years, lung recoil pressure (Pst) at 100%, 90%, and 60% of TLC and static lung compliance (Cst) were measured using an esophageal balloon. The indices of airflow limitation, including maximal expiratory flow at 25% VC ($\dot{V}max_{25}$), forced expiratory volume in 1 second (FEV_1), and specific airway conductance (sG_{aw}), were also measured. In all patients, $\dot{V}max_{25}$ was reduced, the airway obstruction being more pronounced in patients with CF. Pst was reduced in CF and asthma, again more in the patients with CF. Cst was normal in both groups because the pressure-volume curve was shifted up and to the left. There were significant correlations between Pst at 60%, 90%, and 100% of TLC and both $\dot{V}max_{25}$ and FEV_1 ($p < 0.01$). sG_{aw} correlated with Pst90 and Pst60 ($r = 0.47$ and 0.53, respectively; $p < 0.05$ for both). No correlation was found between Cst and Pst at any lung volume. No correlations were observed between Cst and $\dot{V}max_{25}$, FEV_1, or sG_{aw}. These results suggest that loss of elastic lung recoil pressure is a factor in airflow limitation of children and young adults with CF or asthma.

The Relationship of RSV-Specific Immunoglobulin E Antibody Responses in Infancy, Recurrent Wheezing, and Pulmonary Function at Age 7-8 Years—RC Welliver, L Duffy. Pediatr Pulmonol 1993;15:19.

In order to determine if respiratory syncytial virus (RSV)-specific IgE responses at the time of bronchiolitis in infancy are related to recurrent wheezing and pulmonary function at

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[1] Nocturnal Oxygen Therapy Trial Group. Continuous or nocturnal oxygen in hypoxemic chronic obstructive lung disease: a clinical trial. *Ann Intern Med.* 1980; 93:391-8.

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7-8 years of age, a cohort of 43 infants was identified at the time of their initial RSV bronchiolitis episode. RSV-specific IgE responses in nasopharyngeal secretions were determined, and patients were then followed prospectively with notation of the number of wheezing episodes and exposure to cigarette smoke at home. At 7-8 years of age, the patients underwent skin testing to 7 environmental allergens and pulmonary function testing, including pulse oximetry and methacholine challenge. Pulmonary function following inhalation of bronchodilating agents was compared to baseline pulmonary function results in order to determine if abnormalities of pulmonary function were reversible. Recurrent wheezing following bronchiolitis was associated with the initial RSV-IgE response, as well as with a family history of asthma. Current wheezing at age 7-8 years was associated with 2 or more positive skin tests ($p < 0.01$), a history of exercise-induced wheezing ($p = 0.01$), and increased sensitivity to metha-choline ($p < 0.01$). Pulmonary function test results were similar for groups with and without recurrent wheezing following bronchiolitis. For the entire study group, RSV-IgE specific responses were unrelated to pulmonary function, but small airway dysfunction was associated with passive smoking ($p < 0.025$), and both large airway dysfunction and increased airway reactivity were associated with the number of positive skin tests ($p < 0.025$). Reduced small airway function improved following bronchodilator inhalation. The degree of improvement was inversely related to the degree of baseline dysfunction ($p < 0.01$), and was correlated with passive smoking ($p < 0.01$). These findings suggest that decreased pulmonary function following bronchiolitis is related to atopy and passive cigarette smoke exposure and may be reversible.

Acoustic vs Spirometric Assessment of Bronchial Responsiveness to Methacholine in Children—J Sanchez, A Avital, I Wong, A Tal, H Pasterkamp. Pediatr Pulmonol 1993; 15:28.

To study wheezing as an indicator of bronchial responsiveness during methacholine challenge (MC) in children, we used computer analysis of respiratory sounds and compared wheeze measurements to routine spirometry. MC was performed in 30 symptomatic subjects (sympt), age 11 ± 3.1 years (mean ± SD), with suspected asthma and in 12 controls (contr), age 10 ± 3.4 years. Respiratory rate (RR), spirometry, arterial oxygen saturation (S_{aO_2}), and cough were registered until the concentration provoking a ≥ 20% fall in forced expiratory flow in 1 second (FEV_1; PC_{20}), or the end point (8 mg/mL) was reached. For 1 min after each inhalation, sounds over the trachea and posterior right lower lobe were recorded together with calibrated airflow. Computer analysis of respiratory sounds was used for objective wheeze quantification. Wheezing was measured as its duration relative to inspiration (T_W/T_i) and expiration (T_W/T_e). Seventeen of the sympt group developed wheezing (sympt/W) with ≥ 5% T_W/T_i or ≥ 5% T_W/T_e. Thirteen of the sympt did not wheeze (sympt/no W). Three contr developed wheeze (contr/W) while 9 did not (contr/no W). In sympt/W, RR increased from 20 ± 6.2 per min at baseline to 25 ± 9.2 ($p < 0.05$) at the MC concentration provoking wheeze (PC_w), and S_{aO_2} decreased from 97.4 ± 1.2% to 95.3 ± 2.4 ($p < 0.05$). In contr/W, RR did not change, but S_{aO_2} decreased from 97.3 ± 1.5% to 95.7% ± 1.2% ($p < 0.05$). Wheezing occurred at both recording sites and was as common during inspiration as during expiration. In subjects who had both PC_w and PC_{20}, these variables correlated well ($r = 0.82$, $p < 0.0001$). Three SW did not reach PC_{20} (maximum fall in FEV_1 8%, 12%, and 18%). On the other hand, 6 of 13 sympt/no W and 2 of 12 contr/no W had a positive MC. Thus, wheezing during MC had a sensitivity of 68% and a specificity of 82%. Acoustic measurements were reproducible in 14 subjects who performed a second MC within 2-10 months. We conclude that wheezing during MC in children strongly suggests airway hyperresponsiveness: however, wheeze detection cannot fully replace spirometry in bronchial provocation testing.

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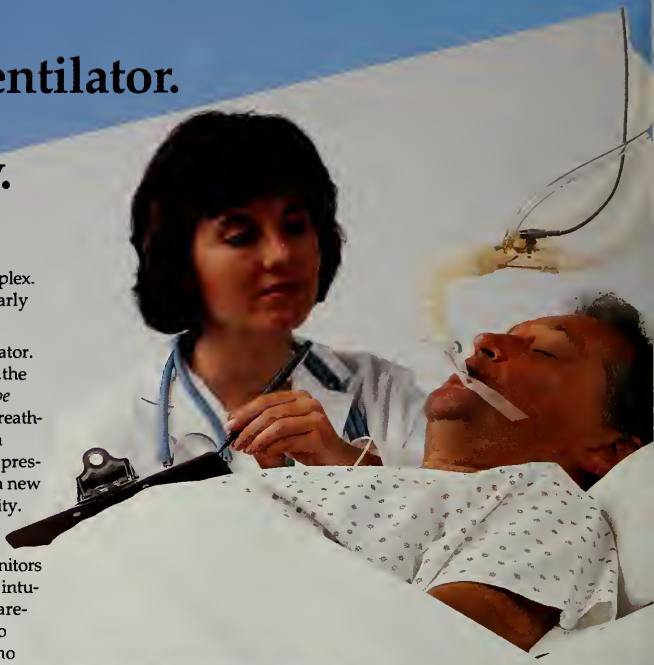

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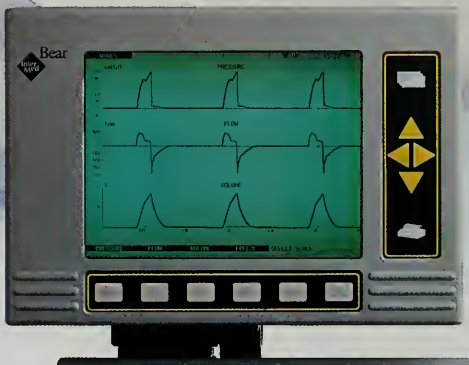

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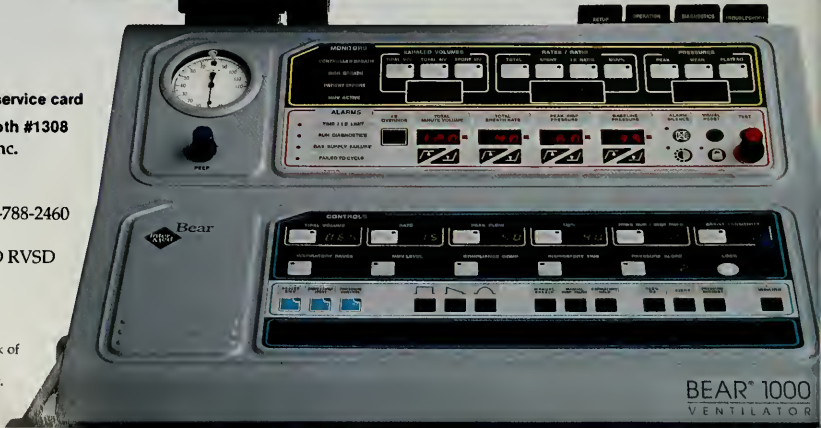

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Humidification of Inspired Gases during Mechanical Ventilation

We have previously addressed the respiratory care community on this editorial's topic, writing both separately[1,2] and together.[3,4] Still, we feel that the importance of humidification during mechanical ventilation remains underappreciated, perhaps because it is seen as playing a supporting role. We also find that myths and misconceptions abound concerning humidification's use. Thus, in this editorial we present some facts on several humidification issues, asking readers to examine, or re-examine, the evidence under the bright light of scientific scrutiny. Five questions are posed: (1) How often should ventilator circuits be changed? (2) What is the role of contaminated condensate in producing nosocomial pneumonia? (3) Do artificial noses reduce the incidence of nosocomial pneumonia? (4) Are all artificial noses created equal? and (5) Can adjusting inspired gas temperature affect body temperature?

How Often Should Ventilator Circuits Be Changed?

In 1983, the Centers for Disease Control called for circuit changes every 24 hours.[5] This is in conflict with the common practice of changing circuits every 48 hours, as suggested by the work of Craven et al in 1982.[6] But what's magical about 48 hours as a limit? More recent papers (1987 and 1991) showed that the time between circuit changes can safely be further lengthened by use of heated wire circuits[7] (changed every 5 days) or artificial noses.[8] However, neither of these studies utilized a control group, causing us to question their conclusions. While we can say that heated wire circuits need be changed only every 5 days, what says that the same is not true for conventional circuits? Dreyfuss and colleagues[9] compared 48-hour circuit changes to no circuit changes and reported in 1991 that the incidence of nosocomial pneumonia was lower when circuits were not changed. They speculated that the less frequently circuits are handled and opened to atmosphere, the less likely it is that contaminants can enter the patient-ventilator system.

Again, the question "How often should ventilator circuits be changed?" begs for an answer. In 1992, Craven et al[10] suggested that 48 hours remains the standard interval for circuits with heated humidifiers, while circuits with artificial noses need never be changed. In a recent study of 120 patients at the University of Cincinnati,[11] we found no contamination of ventilator circuits used for up to 5 days when a hygroscopic condenser humidifier (HCH) was employed in 88 patients. When a heated humidifier (HH) was used in 32 patients, we found that the circuit was contaminated within 48 hours in nearly 70% of cases. However, the incidence of nosocomial pneumonia was similar in both groups—6% in the HH group and 9% in the HCH group—as well as lower than the probably typical 10% reported by Craven et al[10] in 1992. As a result of our study, the published reports cited above, and other reports of safe use of these devices for several days[12-14] (in one instance, 5 days[13]), we have modified our circuit-changing policy. A patient using an HCH never has the circuit changed. A patient using an HH has the circuit changed every 96 hours (4 days), or more frequently if required for aesthetic purposes.

The true answer to the question, then, requires that another question be asked in return: What type of humidification device is being used? Based on the literature to date, there appears to be no reason to change circuits when HCHs are employed. This includes HCHs both with and without filters—because if no condensate is present, no bacteria grow. We believe that patients using HHs can use circuits up to 96 hours until further research is reported. Again, if patients have pulmonary edema or copious secretions and therefore excessively soil their circuits, a policy of more frequent changes can be instituted, tailoring this to the individual patient.

What Is the Role of Contaminated Condensate in Producing Nosocomial Pneumonia?

Contaminated condensate from the ventilator circuit is often considered a culprit in the etiology of nosocomial pneumonia. Equipment manufacturers have reinforced this concept as a strategy to market filters, heated wire circuits, and other specialized circuits and collectors. The implication of contaminated condensate as a cause of nosocomial pneumonia began with a report in 1965 by Reinarz et al.[15] In following years, aerosol generators were determined to be the type of respiratory care equipment capable of introducing pathogens into the bronchial tree.[16-20] Much of this research was done during a period when disposable equipment and supplies were the exception, not the rule, and when nebulizers and humidifiers that produced microaerosols were frequently used. We need to reconsider the role of contaminated condensate in the 1990s.

Where does contaminated condensate come from? Interestingly enough, when tubing condensate is contaminated, the bacteria nearly always originate in the patient.[21-23] Yet somehow it has become commonplace to believe the opposite: that bugs originating in the circuit infect the patient. However, bacteria reaching tubing can incubate there (by virtue of accident or poor technique) and then be lavaged into the airway. The true incidence of pneumonia caused by contaminated condensate is unknown. However, the major cause of nosocomial pneumonia in mechanically ventilated patients is the aspiration of bacteria from the gastrointestinal tract into the airway.[10] Craven et al[10] suggested in 1992 that condensate inadvertently lavaged into the airway during patient movement may result in reinfection of the patient with a higher concentration of bacteria. However, the concept that the incidence of nosocomial pneumonia can be reduced simply by altering respiratory therapy devices is far-fetched. Cross and Roup[24] concluded in 1980 that "contaminated respiratory assistance devices are rarely a direct cause of hospital-associated pneumonia . . .." In this connection, however, it should be remembered that poor technique in the handling of such devices (eg, tubing), as mentioned above, can be a source of trouble. Perhaps an apt analogy here is the warily regarded crate seen at a roadbuilding site, bearing on its side the legend, "This TNT is very safe if handled properly."

This is not an argument for a relaxation of our concern for infection control in the ICU. Rather, it is an attempt at a redirection of efforts. Certainly, condensate should be handled with Universal Precautions, and suction procedures should adhere to strict sterile technique; but purchasing of inspiratory filters and special circuits in an attempt to reduce the incidence of nosocomial pneumonia is economically foolish. The role of condensate in producing nosocomial pneumonia is negligible when disposable circuits with wick humidifiers are used in the absence of aerosol devices and when good technique is used in handling equipment.

Do Artificial Noses Reduce the Incidence of Nosocomial Pneumonia?

An artificial nose may include a filter that 'protects' the patient from pathogens in the circuit. Of course, when an artificial nose is used, there is no condensate, so this is a strange concept. As we have just discussed, nosocomial pneumonia almost always has an endogenous cause, something that an artificial nose cannot prevent.

Interestingly, Gallagher et al[8] reported in 1987 that with use of the Pall HME (heat and moisture exchanger) for a given period, the incidence of *Pseudomonas aeruginosa* "colonization or infection" was 21% (16/75), compared to an incidence of 55% (35/66) with the use of HHs during a similar period 6 months earlier. However, the earlier time period was reviewed retrospectively, and no comparison of the two patient populations was reported. Additionally, the heated humidification system, circuit changes, and other technical details were not described. Perhaps the techniques that were used predisposed the HH systems toward pseudomonas colonization. This paper[8] does not provide enough details to validate the authors' conclusion about the Pall HME.

Reports in the 1990-1992 period by Roustan et al,[21] Martin et al,[22] and Misset et al[23] have compared the incidence of nosocomial pneumonia in patients using the Pall HME to that in patients using HHs. The incidence of nosocomial pneumonia was similar in all three studies, regardless of the

humidification device used. In fact, Martin et al[22] found that circuits were colonized with bacteria in 77% of cases when an HH was used and in 10% when the Pall HME was used. This disagrees with the results of Gallagher et al[8] mentioned above, and further study is needed in order to explain these differences.

Because of the etiology of nosocomial pneumonia, its incidence in all likelihood is not affected by the use of artificial noses per se.

Are All Artificial Noses Created Equal?

Despite the incredible temptation, we resisted calling this section "How To Pick an Artificial Nose." There is, of course, only one answer to that question: very carefully. (Sorry, we can't resist *every* temptation.)

In any event, a myriad of devices are claimed to be "artificial noses." One problem in comparing them is that their moisture output is not recorded systematically and by uniform methodology. Most manufacturers report the moisture output in mg H_2O/L, and then in fine print they detail the test method. This may sometimes be based on a laboratory setup with a tidal volume of 250 mL and a frequency of 20 breaths/min. The device being tested may have a dead space of 90 mL, which means that it probably would never be used in a clinical situation with the test tidal volume and frequency. But, this bench test produces good-looking moisture-output numbers! Then when the device is used clinically and the patient's tidal volume is 800 mL, the moisture output is unknown. In 1990, the International Standards Organization (ISO) reported the development of a standardized model for testing artificial noses that may alleviate this problem.[25,26] The results of this testing have produced findings that were not appreciated by previous workers,[27-29] who had reported peak relative humidity and temperature.

The first of the ISO-standard-based studies[25] has helped to identify the differences between heat and moisture exchangers (HME), which rely solely on physical exchange of heat and water—and hygroscopic condenser humidifiers (HCH), which additionally use a hygroscopic salt to chemically improve heat and moisture exchange. At a tidal volume (V_T) of 1,000 mL and a frequency of 10 breaths/min, the ICOR Triplus HCH delivered 28 mg H_2O/L, compared to 21 mg H_2O/L for the Pall HME. This is particularly impressive when you consider that the ICOR HCH dead space (38 mL) is approximately half that of the Pall HME (87 mL). These different findings for moisture output from these devices contrast vividly with the studies reporting peak humidity and temperature,[27-29] which showed the Pall HME's output to be comparable to that of an HCH. ISO testing standards should be adopted by all manufacturers to create consistent, clinically realistic applied standards and to help clear up the confusion regarding the efficacy of various devices.

Further evidence of the superiority of the HCH over the HME was provided by Bickler and Sessler,[30] who in 1990 reported relative humidity and temperature at the distal end of the endotracheal tube in anesthetized patients when HCHs or HMEs were used. With a V_T of 1,000 mL and a frequency of 10 breaths/min, the use of an HCH resulted in the delivery of an average of 29 mg H_2O/L, whereas the use of an HME yielded 19 mg H_2O/L.

We feel that this is critically important, particularly in light of the studies by Cohen et al,[31] Missett et al,[23] Martin et al,[22] and Roustan et al.[21] All of these studies compared the effect of the Pall HME and HHsS in ICU patients. In each study, occlusion of the tracheal tube occurred regularly when the Pall HME was used. In the work by Martin et al,[22] a patient using the Pall HME died after complete tracheal obstruction, causing the protocol to be terminated. Table 1 reviews the results of these studies.

One problem with all of these studies is that no exclusion criteria for the use of the HME were used. It has been suggested that HMEs should not be used for patients with thick or bloody secretions, core temporatures < 32°C, expired V_T < 75% of inspired V_T, and $\dot{V}_E$ > 10 L/min.[2,4,31] It may be that the above-cited studies[21-23,31] applied HMEs where they would not work effectively or were contraindicated.

At the University of Cincinnati, an algorithm for HCH use was developed and tested in 120 consecutive adult patients in the surgical intensive care unit (SICU) (Fig. 1).[11] Of these patients, 88 were initially treated with an HCH and 32 with an HH. During the study, 10 patients developed exclusion

Table 1. Results of Four Studies Comparing the Incidence of ETT* Obstruction in Patients Using the Pall HME to theIncidence in Patients Receiving Heated Humidification

First Author	Number of Patients HME	Number of Patients HH	Incidence of ETT Obstruction HME	Incidence of ETT Obstruction HH	Author's Conclusions
Cohen et al[31]	170	81	15 (9%)	1 (1%)	HMEs do not provide sufficient airway humidification for generalized ICU use.
Martin et al[22]	31	42	6 (19%)	0	The Pall HME "did not provide sufficient humidification of inspired gas in ICU patients."
Misset et al[23]	30	26	4 (13%)	2 (8%)	HME should be replaced if difficulty in suctioning occurs.
Roustan et al[21]	155	61	9 (16%)	0	Efficiency of the Pall HME may be low in some conditions of ambient humidity.

ETT = endotracheal tube, HME = heat & moisture exchanger, and HH = heated humidifier.

criteria for continued HCH use, and HHs were substituted. The use of the algorithm resulted in safe and efficient humidification in all patients, and no tracheal tube obstruction was seen.

We caution that the use of this algorithm may provide different results in other ICUs, where patients may be different than in an SICU. Nevertheless, we believe that use of the algorithm will still result in safe application of the appropriate device. We also caution against using an HME alone, because of the high incidence of tracheal tube obstruction. Based on our review of the literature, we believe that the only safe use of the Pall HME in the ICU requires the addition of a cold-water-bath humidifier, as described by Suzukawa et al.[32]

Can Adjusting Inspired Gas Temperature Affect Body Temperature?

To address this question, first we must appreciate the thermodynamics of body-temperature regulation. Basal metabolic heat production is es-

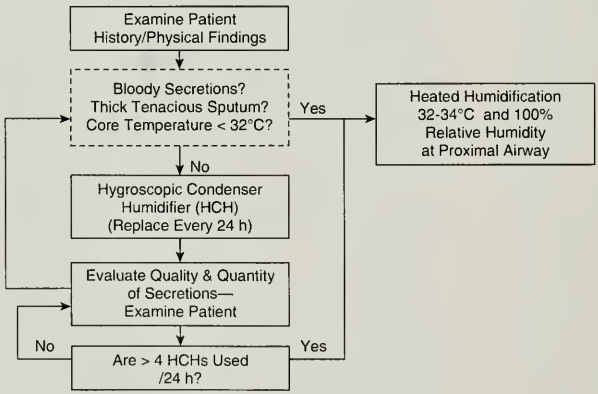

Fig. 1. University of Cincinnati algorithm for the application of a hygroscopic condenser humidifier (HCH) and/or a heated humidifier.

timated to be about 46 watts/m^2 of body surface area (BSA).[33] An adult weighing 68 kg and 74 cm tall has a BSA of about 1.8 m^2, so his heat production would be about 82.8 watts (W). (Note: 1 kilocalorie/h = 4.19 joules/h = 1.6 W.[34]) It follows that at steady state, the body must lose heat to the environment at the same rate that heat is produced—in order to maintain a constant body temperature of 37°C. Heat loss occurs through radiation (60% of total heat loss in adults), conduction (3%), convection (12%), and evaporation (22%).[33] Heat loss from the airways amounts to about 17 W, or only about 21% of total heat loss.[35] Most of the heat transfer from the airways is through evaporative (insensible) heat loss, with the balance due to convective (sensible) heat loss.

Respiratory heat exchange is a function of minute ventilation, gas density, specific heat, latent heat of vaporization, water vapor content, and temperature gradient,[36] but, basically, heat exchange is proportional to minute ventilation and either temperature or water vapor content gradients (ie, inspiration vs expiration).

For convective heat exchange:

$$\dot{W}_{CV} = \dot{V}\rho c_p (T_E - T_I),$$

where $\dot{W}_{cv}$ = convective heat exchange, $\dot{V}$ = minute ventilation, ρ = gas density, c_p = specific heat of gas, T_E = expired gas temperature, and T_I = inspired gas temperature;

and for evaporative heat exchange:

$$\dot{W}_{EV} = \dot{V}\lambda\ (C_{E\text{-}H_2O} - C_{I\text{-}H_2O}),$$

where $\dot{W}_{EV}$ = evaporative heat exchange, $\dot{V}$ = minute ventilation, λ = latent heat of water evaporation, $C_{E\text{-}H_2O}$ = water vapor concentration of expired gas, and $C_{I\text{-}H_2O}$ = water vapor concentration of inspired gas.

In these equations, gas density, specific heat, and volume are assumed to be the same for inspired and expired gas.

Figure 2 illustrates the temperatures and water vapor contents of inspired and expired gas during normal breathing. Assume that an intubated patient's respired gas conditions approximate those of tracheal gas (because the upper airway is bypassed). From Figure 2 and the equations above, we see that when inspired gas temperature is > 22°C and water vapor content is > 9.7 mg/L, normal heat loss is reduced. When inspired gas temperature reaches 37°C and water vapor content reaches 43.9 mg/L, heat loss from the airways is eliminated. If temperature and water vapor content rise further, the airways experience a net heat *gain*.

Inhalational rewarming as a field treatment for hypothermia was introduced in 1972 by Lloyd et al.[37] Subsequent studies provided evidence both for[38-41] and against[42,43] its use. Differences in experimental protocols and body-temperature measurement probably account for the disagreements.[44] Aware of the problems, Romet and Hoskin[44] compared temperature and metabolic responses to inhalational and water-bath rewarming protocols. Volunteer subjects were cooled in a whole-body calorimeter controlled at 22°C. (They were breathing spontaneously, but findings from the study can be extrapolated to conditions of mechanical ventila-

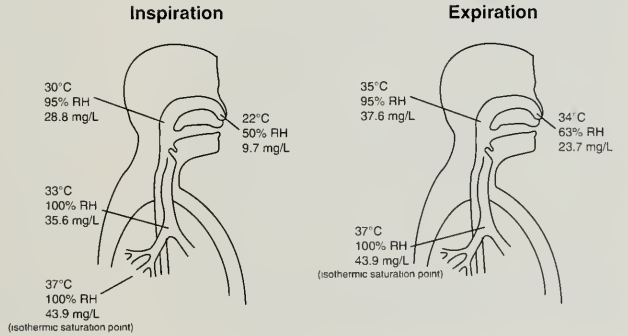

Fig. 2. Temperatures and water vapor contents of inspired gasand expired gas during normal breathing. See text for application to an intubated patient.

tion.) Rewarming to a temperature of 35.6-36.9°C was accomplished by (1) shivering under wool blankets (as a control), (2) inhaling humidified air at either 40 or 45°C, or (3) bath rewarming in water controlled at 40°C. There were no significant differences in rewarming rates between shivering and either of the inhalation protocols. While inhalation of heated humidified air provided a net heat gain, it was small (< 7%) compared to the total heat gained during the rewarming period. Furthermore, breathing heated air caused a reduction in metabolic rate and ventilation that was not compensated for by the heat gained through respiratory heat exchange. This confirms earlier data from work by Morrison et al,[41] who calculated a reduction of 1.4 kJ of metabolic heat for every 1.0 kJ of respiratory heat added. Romet and Hoskin[44] speculated that the breathing of warm air suppresses laryngeal cold receptors (which have an upper threshold of 36-40°C[45]) and hence decreases ventilation and metabolic rate. As might be expected, the inhalation of heated and humidified air was "not well tolerated by some of the subjects."

If we define optimal inspired temperature as that which maintains normal physiologic conditions (ie, keeps the isothermal saturation point at the normal location in the airway during inspiration), gas delivered to intubated patients should have a temperature of about 33°C and a relative humidity of about 100%.[46,47] It appears that raising the inspired gas temperature a few degrees with the hope of speedy rewarming contributes, at best, little to heat exchange; at worst, it may slow the rewarming by decreasing the patient's metabolic rate. It may also be uncomfortable for the patient. On the other hand, decreasing inspired gas temperature to < 36°C may also be counterproductive because it may (1) contribute little to total body heat loss, (2) increase the risk of mucus-thickening and airway obstruction, and (3) possibly stimulate cold receptors that might increase metabolic heat production.

To answer our question, then, current data indicate that body temperature cannot be efficiently controlled by manipulating inspired gas temperature. That temperature should therefore be maintained at 33 ± 1°C for intubated patients, and efforts to optimize body temperature should be made by other means.

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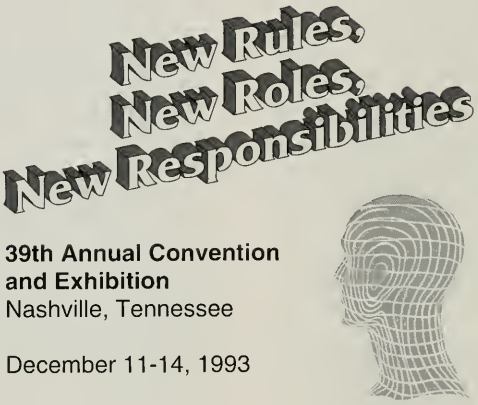

39th Annual Convention and Exhibition
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Analysis of an Endotracheal Intubation Service Provided by Respiratory Care Practitioners

Janice J Thalman BS RRT, Susan Rinaldo-Gallo MEd RRT, and Neil R MacIntyre MD

Our Respiratory Care Services Department provides an endotracheal intubation service that responds to all intubation requests. Intubation is performed by registered respiratory therapists who complete an 8-hour training program, advanced cardiac life support (ACLS) training and certification, and clinical performance of intubation with supervision. The goals of this service are (1) to provide competent persons for performing this service, (2) to assure a rapid response time, and (3) to be cost-effective. EVALUATION METHODS: A retrospective analysis of our service was conducted over a 1-year period (7/90 to 6/91), and calculations were made of the intubation success rate and complication rate. RESULTS: Of the 833 total intubations, 791 were successfully performed by respiratory care practitioners; 730 of those successful intubations (92.3%) were accomplished in fewer than 3 attempts. Recognized complications occurred in 96 intubations (12.1%) and included oral bleeding, vomiting, and short periods of oxygen desaturation. In the 5.1% (42) of the patients not intubated by our service, 22 required heavy sedation, and an anesthesiologist was consulted; 17 patients were intubated by other physicians; and 3 tracheotomies were performed. Multiple intubation attempts were a result of secretions, induced bradycardia, blade-light malfunction, damaged cuff, and esophageal intubations. CONCLUSION: Respiratory Care Services can provide an effective intubation service. Cost savings were realized by centralizing equipment. (Respir Care 1993; 38:469-473.)

Introduction

Emergency endotracheal intubation may be necessary for the management of airway compromise, respiratory insufficiency or failure, or full cardiopulmonary arrest. Acute care hospitals must be capable of providing this service in a rapid, effective, and safe fashion. Traditionally, this service has been provided by physicians, especially anesthesiologists, who are on call. However, data suggest that properly trained nonphysicians can also provide an effective emergency airway service. These data come from experience both with respiratory care practitioners in the hospital[1-3] and paramedics in the out-of-hospital setting.[4] Moreover, it has been suggested that in the hospital setting, respiratory care practitioners can provide this service in a more timely and cost-effective manner.[1-3,5] Further, spokesmen for the respiratory care profession have advocated intubation and Advanced Cardiac Life Support (ACLS) training for therapists involved in critical and emergency care.[6,7]

Our institution has utilized the Respiratory Care Services Department to provide emergency airway support (including intubation) for the last 27 years. This service uses personnel who have received specialized training in intubation that includes 8 hours of formal instruction. Course content includes technique, indications, complications, pharmacology, and medical-legal issues. Written exams and prac-

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tical simulation exams follow the training course. Practitioners must then perform 10 clinical intubations with a supervisor. Practitioners selected for intubation training must be registered respiratory therapists and must have satisfactorily completed instruction in ACLS. Pediatric practitioners are required to have satisfactorily completed instruction in Pediatric Advanced Life Support. The staffing of Respiratory Care Services in our institution is such that 4 to 6 practitioners are available at any given time to provide coverage for a 9-story, 1,000-bed hospital. Skills maintenance is assumed because of the high volume of procedures performed by this centralized service.[8] We believe this centralized approach to be cost-effective because nonphysicians are the primary providers and equipment is centralized (ie, emergency airway equipment is handled only by the respiratory care airway team).

Evaluation Methods

The performance of our intubation service was assessed through a retrospective analysis of all intubation records (Fig. 1) over a 1-year period (July 1, 1990, to June 30, 1991). Records were reviewed for indications, intubation success rate, intubations successfully performed in less than 3 attempts, and complication rate.

Results

Respiratory Care Services received 963 intubation requests during the study period. Assessment following response revealed that immediate intubation was not needed in 130 cases. The majority of these patients were only bag-mask ventilated. One received a treatment with racemic epinephrine, one was nasotracheally suctioned, and for one "Do Not Resuscitate" had been specified.

The remaining 833 patients were successfully intubated—791 (94.9%) of these by Respiratory Care Services and the remainder by physicians. Of the successful intubations 222 (28%) were performed during cardiopulmonary arrest. In 730 (92.3%) of the intubations, fewer than 3 attempts were made (our usual standard before backup help is requested). Reasons for multiple attempts were secretions, induced bradycardia, blade-light malfunction, damaged cuff, and esophageal intubation.

Complications were documented in 96 of the intubations performed by the respiratory care service. The most common complications were oral bleeding, vomiting, and periods of oxygen desaturation. Serious complications requiring intensive medical or surgical therapy were not documented in any of the intubations.

Of the 42 patients in whom intubations could not be completed by Respiratory Care Services, 22 required heavy sedation and/or neuromuscular blockade and were ultimately intubated by an anesthesiologist. Tracheotomies were performed in 3 patients. The other 17 patients were intubated by physicians other than anesthesiologists.

Discussion

Our retrospective 1-year review demonstrates that a respiratory care service can provide effective endotracheal intubation services. Our 94.9% success rate agrees with other reports showing that properly trained nonphysicians can perform appropriate emergency airway management.[1-5] Our complication rate (12%) is also comparable to rates in other reported studies.[9,10]

Several additional features of a centralized respiratory care emergency airway team merit discussion.

(1) A specialized team, strategically located, can respond to an emergency rapidly with all necessary equipment. This rapid response is a consequence of using respiratory care personnel who are already performing clinical duties in the hospital instead of relying on physicians who may be responsible for operating rooms or emergency rooms, or who may be outside the facility.

(2) Centralization of portable emergency equipment is cost-effective, avoids loss, assures optimal function, facilitates compliance with infection control guidelines, and guarantees proper maintenance. In our institution, the total cost for eight portable emergency bags carried by the respiratory care airway team is $24,000 (Fig. 2). The initial expense of providing this equipment for all of the emergency carts throughout the medical center (150 total) would be $450,000.

DUKE UNIVERSITY MEDICAL CENTER
RESPIRATORY CARE SERVICES
Airway, Standby, and Extended Care Record

DATE: TIME:

LOCATION: _____

CODE:

☐ Emergency Airway ☐ Airway Standby/Trach Change ☐ *Extended Care X _____ ☐ Vent Drive Assessment

☐ Airway Management ED ☐ Airway Management Transport ☐ Airway Management Infant Stabilization

REASON/INDICATIONS:

☐ Route for Mechanical Ventilation ☐ Respiratory Distress ☐ Infant Stabilization
☐ Cardioversion/Cardiac Instability ☐ Bronchoscopy ☐ Sedation
☐ Code 5 on Intubated Patient ☐ Weaning Trach ☐ Trauma
☐ Code 5 ☐ Other _____

ACTIONS TAKEN:

☐ Breath Sounds Confirmed ☐ CXR Ordered ☐ Bag/Mask Ventilation ☐ Anesthesia Called:
☐ Visual Observation ☐ Trach Change ☐ Pulse Oximetry ☐ MD Request
☐ Oxygen Administration ☐ Vent Transport ☐ NG Tube Insertion ☐ Sedation
☐ End Tidal CO_2 Confirmed ☐ ET Tube Change ☐ Mechanical Ventilator ☐ Unable to Place ET tube
☐ Other _____

PROCEDURES:

☐ Intubation: ☐ Nasal: Naris L R ☐ Oral : Blade/Type _____
# of Attempts _____ # of Visualizations _____ Tube Size _____
Placement _____

☐ Trach Change: From: Size/Type _____
To: Size/Type _____

COMPLICATIONS: ☐ None ☐ Unable to Place ☐ Oral/Nasal Bleeding ☐ Vomiting/Aspiration
☐ Tooth/Jaw Damage (specify) _____
☐ Other (specify) _____
☐ Physician Notified (name) _____

MEDICATIONS: ☐ None ☐ _____

COMMENTS: _____

Assistant: _____ **Assistant:** _____

PRACTITIONER SIGNATURE: _____

REVIEWED BY: (SUPERVISOR/TEAM LEADER) _____

* 1 Extended Care = 30 Minutes

Fig. 1. Clinical record completed for each attempted intubation. Study data were compiled from such records.

Emergency Airway Box Inventory (Adult)
Respiratory Care Services

- ___ 1 Laerdal Bag
- ___ 1 Reservoir
- ___ 1 Mask
- ___ 1 Oxygen Tubing
- ___ 1 Partial Rebreather
- ___ 1 O_2 Flowmeter with Wing Nut
- ___ 2 BioChem Adapters + Tubing
- ___ 1 Magill Forceps (adult)
- ___ 1 Yankauer Suction Tip
- ___ 2 Stylets
- ___ 2 Laryngoscope Handles
- ___ 2 Phillips #2 Blades

MacIntosh Blades
- ___ #3
- ___ #4

- ___ 1 Wisconsin Blade (#4)
- ___ 1 Hollister Spray
- ___ 1 Pink Tape
- ___ 2 10-mL Syringes
- ___ 4 Large Bulbs for Laryngoscope
- ___ 2 Size C Batteries

ET Tubes—
- ___ 7.0
- ___ 7.5
- ___ 8.0 (2)
- ___ 8.5 (2)
- ___ 9

Trigger Tubes—
- ___ 7.0
- ___ 8.0
- ___ 9.0

Airways—
- ___ 2 Small
- ___ 2 Medium
- ___ 2 Large
- ___ 1 Luomanmen Airway

Jet ET Tubes
- ___ 8.0
- ___ 9.0

- ___ 1 Introducer
- ___ 1 Racemic Epinephrine (unopened)
- ___ 1 Cetacaine Spray
- ___ 1 Xylocaine Jelly
- ___ 1 BioChem 515

Date _____ Checked by _____

Fig. 2. Inventory checklist for Respiratory Care Services Adult Emergency Airway Box.

(3) Utilization of nonphysicians to provide emergency airway coverage is a more efficient use of health care personnel because it allows physicians to remain at their primary duties and be utilized only for backup support.

(4) Centralization of an intubation service can provide accurate records of intubation data and emergency situations for the institution (Fig. 1). This can provide valuable information on current practice and procedure, quality concerns, and legal questions.

(5) An emergency airway team gives high visibility to the respiratory care department, which improves morale and recruitment.

Conclusion

We conclude that properly trained respiratory care practitioners can provide an emergency airway service that is not only safe but is also cost-effective.

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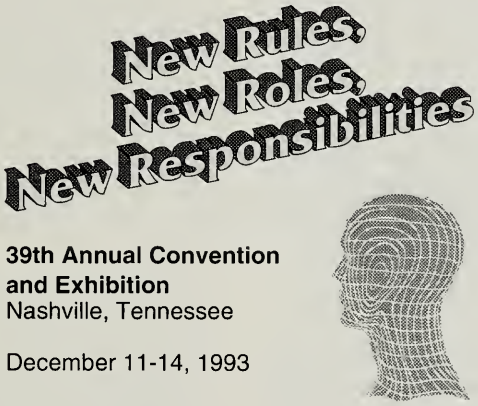

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Effects of In-Line Nebulization on Preset Ventilatory Variables

Usama Hanhan MD, N Kissoon MD, M Payne RRT, C Taylor RRT,
S Murphy PhD, and LK DeNicola MD

BACKGROUND: The effects of nebulization on preset ventilator variables have not been fully studied. The objectives of this study were to determine the effect of in-line nebulization on exhaled volume (V_E), peak inspiratory pressure (PIP), and positive end-expiratory pressure (PEEP) during ventilation, and to compare these effects among three ventilators (Bear Cub, Sechrist Infant, and Siemens 900C). METHODS & MATERIALS: In an in-vitro experiment, we used ventilator circuits attached to a model-test lung and five groups of ventilator variables to simulate clinical situations. The ventilator circuits were connected to an infant test lung; a pressure probe and respirometer were connected at the expiratory limb; and a small-volume nebulizer was attached in the inspiratory limb of the circuit. Baseline measurements and changes in PIP, V_E, and PEEP obtained during nebulization were repeated in triplicate in the pressure-controlled mode of the Bear Cub and Sechrist Infant and in the pressure- and volume-controlled modes of the Siemens 900C. Results were analyzed using repeated measures of variance with $p < 0.05$ considered statistically significant. Newman-Keuls multiple range test was used for post-hoc comparison procedures. RESULTS: Statistically significant changes were observed in mean V_E and PIP during nebulization in all ventilators studied ($p < 0.01$). PEEP increased by 1-2 cm H_2O in the Sechrist Infant ventilator during nebulization. Changes from baseline were consistent across all five ventilator settings tested. CONCLUSIONS: In-line nebulization results in changes in V_E and PIP. The least possibly adverse changes were seen with the Bear Cub and Sechrist Infant ventilators. To avoid potential complications, preset ventilator variables may need to be adjusted when in-line nebulization is performed. (Respir Care 1993;38:474-478.)

Background

Aerosolized bronchodilators are among the most commonly offered respiratory therapies in critical care units. Delivery of medication by aerosol has the advantage of delivering high concentrations of a drug to the lungs while minimizing undesirable systemic side effects.[1] In the mechanically ventilated patient, these medications are often aerosolized by a jet nebulizer placed in the inspiratory limb of the ventilator circuit.[2] Despite the potential for possible distortion of the preset pressures and volumes, few reports have been published on the effects of such nebulization on the preset ventilator variables.

Changes in the delivered pressures and volumes could result in under- or overventilation that might be detrimental to the patient with compromised pulmonary function. In addition, unrecognized changes in positive end-expiratory pressure (PEEP) or peak inspiratory pressure (PIP) might lead to further morbidity such as barotrauma and air leak syndromes. Our personal experience suggested that the addition of in-line nebulization results in alteration of the preset PIP, PEEP, and exhaled volume (V_E). However, these changes were inconsistent and varied with the type of ventilator and the settings used.

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Cognizant of the deficiencies in the literature, we used a lung model to determine the effects of nebulization on the PIP, PEEP, and V_E, and to compare the effects seen with three different ventilators commonly used in our institution.

Methods and Materials

Three ventilators (the Siemens 900C,* Bear Cub, and Sechrist Infant ventilator) were studied. All ventilators were tested in the pressure-controlled mode. In addition, the Siemens 900C was also studied in the volume-controlled mode. As outlined in Figure 1, the ventilators were connected to central oxygen and air sources and electrical power in the usual manner. An Infant Star test lung was attached at the patient-connector site of the ventilator circuit. Pressure changes were measured using an in-line pressure probe on the expiratory limb. Exhaled volumes were measured using a Wright respirometer placed in-line on the expiratory limb of the ventilator circuit. An Airlife Misty Neb nebulizer was attached at the inspiratory limb of the circuit.

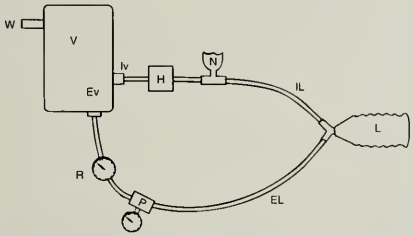

Fig. 1. Circuit diagram. V, ventilator; Iv, inspiration valve; H, heated humidifier; N, nebulizer; IL, inspiratory limb; L, test lung; EL, expiratory limb; P, manometer; R, Wright respirometer; Ev, exhalation valve; and W, wall O_2/air.

Prenebulization

The ventilator circuits were connected as described and then cycled at preset variables as outlined in Table 1. The ventilator settings were chosen to reflect those commonly used in disease states. For example, patients with asthma or bronchiolitis might require low PEEP and low respiratory rates, whereas a patient with adult respiratory distress syndrome (ARDS) would require higher levels of PEEP. Prior to in-line nebulization, baseline measurement of PIP, V_E, and PEEP were obtained in triplicate and recorded.

Table 1. Pressure-Controlled Ventilator Settings Used in Study of Effect of Continuous In-Line Nebulization

Setting	PIP* (cm H_2O)	PEEP (cm H_2O)	Rate (breaths/min)	Inspiratory Time†
1	20	4	20	0.25
2	25	6	25	0.30
3	30	8	30	0.35
4	35	10	35	0.40
5	40	12	40	0.45

*PIP = peak inspiratory pressure; PEEP = positive end-expiratory pressure.
†Fraction of respiratory cycle time.

Nebulization

We elected to test normal saline (NS) and albuterol as our nebulized solutions. NS was tested separately because it is a common vehicle for most nebulized medications. The effects of jet nebulization of 3 mL of NS at a flowrate of 6 L/min were examined at each of the five ventilator settings outlined in Tables 1 and 2. NS was nebulized through the ventilator circuit for 5 minutes to allow for stabilization of all variables. While nebulization continued, values for V_E, PIP, and PEEP were recorded at 2-min intervals for a total of three readings. Total nebulization time was 11 minutes. Fol-

Table 2. Volume-Controlled Ventilator Settings Used in Study of the Effect of Continuous In-Line Nebulization

Settings	Tidal Volume (mL)	PEEP (cm H_2O)	Rate (breaths/min)	Inspiratory Time*
1	50	4	40	0.20
2	100	6	35	0.25
3	150	8	30	0.33
4	200	10	25	0.43
5	250	12	20	0.50

*Fraction of respiratory cycle time.

*Suppliers are identified in the Product Sources section at the end of the text.

lowing re-establishment of baseline values, albuterol (2.5 mg in 3 mL NS) was tested in the same manner outlined above.

Table 3. Mean Exhaled Volumes (in L/min) at Baseline and with Nebulization*

	Baseline	Albuterol	Change from Baseline
Siemens, Volume Control	4.8 (1.4)†	9.6 (1.8)	97%
Siemens, Pressure Control	3.0 (1.4)	7.7 (1.1)	150%
Bear Cub	15.3 (0.5)	22.7 (0.2)	50%
Sechrist	15.4 (0.6)	23.3 (1.6)	49%

*Data from saline trials were not statistically different from albuterol trials.
†Values are expressed as mean (SD).

Values for the measured variables were averaged. Because there were no differences in outcome between the NS and albuterol trials, only albuterol data are presented in Tables 3 and 4. Mean changes from baseline in PIP and V_E were analyzed using repeated measures analysis of variance with $p < 0.05$ considered statistically significant. Newman-Keuls multiple range test was used for post-hoc comparison procedures.

Table 4. Mean Peak Inspiratory Pressures (cm H_2O) at Baseline and with Nebulization*

	Peak Inspiratory Pressures		Change from
	Baseline	Albuterol	Baseline
Siemens, Volume Control	59.6 (31.3)†	70.7 (31.8)	19%
Siemens, Pressure Control	30.2 (7.4)	45.7 (9.6)	+53%
Bear Cub	30.1 (7.7)	28.1 (8.1)	–6%
Sechrist	30.0 (7.9)	31.6 (7.4)	+5%

*Data from saline trials were not statistically different from albuterol trials.
†Values are expressed as mean (SD).

Results

A total of 180 observations was made among all three ventilators. The mean PIP and V_E prior to and during nebulization are shown in Tables 3 and 4. A statistically significant increase ($p < 0.001$) was seen in mean exhaled volumes during nebulization in all three ventilators (Table 3). Using Newman-Keuls multiple range test as a post-hoc comparison procedure, we found a significant difference between baseline and the nebulized solution ($p < 0.01$). The magnitude of change, however, was larger on the Siemens 900C in both volume- and pressure-controlled modes.

Mean PIPs were also significantly different during nebulization on all ventilators (Table 4). Again, significant differences existed between baseline and the nebulized solution ($p < 0.05$). It is important to note that while changes in pressure were statistically significant for all ventilators, the magnitude of change varied greatly. The 5-6% change found for the Bear Cub and Sechrist is probably not clinically relevant, although statistically significant. PEEP increased by 1-2 cm H_2O during nebulization using the Sechrist Infant ventilator.

Discussion

Administration of aerosolized medications through the ventilator circuit is commonly used to treat intubated patients with various pulmonary conditions. This route of administration has gained particular attention in recent years as one of the mainstays of therapy for patients in status asthmaticus. It also has been found useful in treating patients with bronchopulmonary dysplasia and bronchiolitis.[3-5]

Several reports have addressed the efficiency, delivery, and dosing of bronchodilators administered to intubated patients during mechanical ventilation.[6-8] Others also have investigated the efficiency of aerosol deposition to the lung, comparing deposition from a metered dose inhaler (MDI) and that from a jet nebulizer in ventilated patients.[2,9] However, reports on the effect of jet nebulization of various solutions on the preset ventilatory variables are scarce in the literature. Such changes, if unrecognized, may lead to deleterious effects, including hypo- or hyperventilation and increased risk of barotrauma.

In our study, we investigated the effects of in-line aerosol nebulization during ventilation on the preset ventilator variables. We selected these variables because these settings may be used to pro-

vide ventilatory support in the treatment of diverse pulmonary pathologies (for example, air trapping in asthma and bronchiolitis and low lung volumes as seen in ARDS and pneumonia).

While the V_E was measured at the expiratory limb and hence represents the V_E of the circuit, the compliance of the test lung (1 mL/cm H_2O) was low and hence the differences in V_E will be small. Our results showed significant increases in V_E in all ventilators studied ($p < 0.001$). These changes may be partially explained by the extra flow that was added during the process of nebulization. The changes occurred irrespective of the ventilator settings utilized, also suggesting that they were the result of the excess 6 L/min added to the circuit during nebulization. Undesirable excess volumes, if unrecognized and uncorrected, may result in overinflation, hyperventilation, and increased PEEP and PIP. These factors may contribute to barotrauma and decreased venous return in patients with minimally adequate intravascular volume. In our model, we demonstrated an increase in PIP and a minimal increase in PEEP. While all changes might be clinically significant and undesirable, the magnitude of change in exhaled volumes was more pronounced on the Siemens 900C in both volume and pressure modes. Changes of this magnitude have been previously reported during nebulization through the circuit of a Servo 900C ventilator using an infant test lung.[10]

PIP was also altered in all three ventilators. The magnitude of change was least in the Bear Cub and Sechrist Infant ventilators. The observation that PIP was altered less in the Bear Cub (6%) and Sechrist Infant ventilators (5%) than in the Siemens 900C pressure control (53%) was particularly interesting. This difference may be accounted for by the location of the pressure-limit valve on these ventilators. The pressure-limit function of the Bear Cub and Sechrist infant ventilators is integral to the exhalation valve.[11] Accordingly, when extra flow is added, such as that from the jet nebulizer in the inspiratory limb of the circuit, it will be detected and accounted for by the pressure-limit function of the ventilator at the expiratory valve (Fig. 1). In contrast, the inspiratory valve of the Siemens 900C regulates the inspiratory gas flow and pressure with each ventilator cycle.[12] The jet nebulizer is attached at the inspiratory limb of the circuit distal to the humidifier and hence bypasses the inspiratory valve regulatory function. Therefore, the increased flow in the Siemens 900C circuit is not detected by the inspiratory valve regulatory mechanism. Our data are supported by findings of previous investigators showing significantly greater changes in PIP during small-volume ventilation of an infant lung model using the Servo 900C.[10] The magnitude of change in PIP was statistically significant for all ventilators; however, the magnitude of changes seen with the Bear Cub and Sechrist is small and may not be clinically important.

During nebulization, we noticed an increase in PEEP from baseline of 1-2 cm H_2O with the Sechrist ventilator. This observation was consistent across all five settings tested and suggests that the changes were a result of the excess of 6 L/min flow in the circuit. Changes of that magnitude may not pose a problem for most patients. We feel, however, that it is important for health care personnel to be aware of this phenomenon because such changes in PEEP pressures may adversely affect patients already on high PEEP, particularly those with low lung compliance and minimally adequate intravascular volume.

In addition to the changes in PIP, PEEP, and V_E that occur during in-line nebulization, other authors have reported adverse effects of continuous in-line nebulization on pressure support ventilation. Using a lung model, Beaty and associates[13] studied the effects of continuous in-line nebulization on the Siemens 900C pressure support mode. They concluded that the use of continuous in-line nebulizers may seriously impair a patient's ability to trigger the ventilator in the pressure support mode.[13] Similarly, Salyer and associates, using a pediatric lung model, have suggested that overdistention of the lung is possible if care is not taken when the Siemens 900C is used during nebulization.[10] Although published experience has been limited to two case reports in adult patients[13] and an in-vitro study,[10] we believe that their results are relevant to the child. Inability to trigger the ventilator would seriously impair ventilation and oxygenation and might lead to atelectasis.

Our results reflect the effects of in-line nebulization on ventilator variables in a model test lung with low compliance. Furthermore, there was no leak at the ET-tube (as might be found with an uncuffed tube), and only a few variables were tested. Therefore, the changes from baseline measure-

ments reported in Tables 3 and 4 were not meant to be used as absolute guidelines in clinical practice but rather to illustrate that significant changes do occur. Another important factor not addressed by our study is the effect of ventilator design or mode on the delivery of drugs to the patient's airway. While a discussion of this subject is beyond the scope of this report, it would seem that continuous-flow systems such as the Bear Cub and Sechrist might be associated with dilution of the drug. In addition, more drug may bypass the airway with these systems. These factors obviously warrant further investigation. In addition, whether changes in ventilator variables during nebulization of the magnitude we found in our model lung do exist in clinical practice is yet to be determined.

Conclusions

Based on our results, we believe that health care personnel in pediatric critical care units need to be aware that in-line nebulization, when administered through the ventilator circuit, results in an increase in the preset PIP and V_E. Although any changes in V_E and PIP might be undesirable, our results showed that the least changes occur in the Bear Cub and Sechrist Infant ventilators when an in-line nebulizer is added to the circuit. To avoid potential complications, we believe that the desired ventilator variables need to be checked and adjusted whenever an in-line nebulizer is attached to the ventilator circuit, irrespective of the type of ventilator used.

ACKNOWLEDGMENTS

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PRODUCT SOURCES

Ventilators:
Bear Cub BP 2001, Bear Medical Systems, Riverside CA
Sechrist Infant Model IV-100B, Anaheim CA
Siemens 900C, Siemens-Elema, Solna, Sweden

Test Lung:
Infant Star, Infrasonics Inc, San Diego CA

Nebulizer:
Airlife Misty Neb, Baxter Healthcare Corp, Valencia CA

Pressure Probe:
Sechrist 1705, Electronic Manometer, Sechrist, Anaheim CA

Respirometer:
Wright Respirometer, Medi-Mold Enterprises Inc, Vernon CT

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Pulmonary Edema Induced by Endotracheal Tube Obstruction: A Case Report

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Introduction

Pulmonary edema is one of the most serious complications in anesthetic practice. Recently, pulmonary edema associated with negative airway pressure caused by acute upper airway obstruction has been identified.[1,2] This negative-pressure pulmonary edema (NPPE) may be the result of epiglottitis, laryngospasm, airway tumor, or strangulation.[2-7] The pathogenesis of NPPE, which is believed to be triggered by extreme transpulmonary pressure gradients, is not completely understood. We describe a case of NPPE secondary to endotracheal tube obstruction caused by a patient's biting the tube while awakening from general anesthesia.

Case Summary

A 16-year-old boy weighing 72 kg was admitted for outpatient surgery to undergo skin grafting for burn-scar contracture of the forearm and wrist. He had been in good health and was taking no medication. Two previous skin graft surgeries under general anesthesia had been completed uneventfully. No abnormal findings were detected in the preoperative physical and laboratory examinations or on review of the chest radiograph. Anesthesia was induced with thiopental 350 mg, followed by intravenous admini-stration of succinylcholine 80 mg. The trachea was intubated orally with an 8.0-mm cuffed endotracheal tube. No other muscle relaxant was given during the procedure. Anesthesia was maintained with nitrous oxide and isoflurane in oxygen and intravenous fentanyl (total dose 300 μg; last dose 5 min prior to surgical incision). The ventilation was controlled from the induction of anesthesia to the end of surgery. The arterial blood pressure and pulse rate remained stable. Oxygen saturation was maintained at 98-99% and expiratory end-tidal CO_2 at 37-48 torr. The surgical procedure was completed uneventfully 110 minutes after the induction of anesthesia. Intraoperative blood loss was 75 mL, and intravenous infusion volume was 1,350 mL.

Immediately after the completion of the procedure, anesthetic gases were discontinued, and 100% oxygen was provided; the patient was still breathing spontaneously through the endotracheal tube. Upon awakening from anesthesia, the patient became physically agitated, flailing his extremities furiously. He did not respond to spoken command. In this state, he bit down hard on the endotracheal tube and made vigorous inspiratory efforts against the obstructed tube, with marked abdominal and supraclavicular retractions. Attempts to insert an oral airway as a bite-block failed, and he could not be ventilated effectively for approximately 3 minutes. His oxygen saturation decreased from 98 to 65%. An oral airway was finally placed successfully, making ventilation possible. Naloxone (0.1 mg) was administered, and the endotracheal tube was then removed.

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As the patient was extubated, a small amount of pink frothy secretion issued from the endotracheal tube. Moist rales were heard in both lung fields, but the respiratory pattern and effort were normal. He was transferred to the recovery room with supplemental oxygen. Analysis of arterial blood gas levels revealed pH of 7.37, P_{CO_2} of 48 torr, and P_{O_2} of 54 torr, with 100% oxygen delivered by simple face mask. (Delivered oxygen concentration can only be estimated at approximately 55% with this delivery system.[8]) He expectorated small amounts of pink frothy sputum frequently. The chest radiograph disclosed bilateral fluffy infiltrates consistent with pulmonary edema. A tentative diagnosis of NPPE was made.

He remained fully awake and was asymptomatic except for his productive cough. Auscultatory and chest x-ray findings taken 5 hours after arrival in the recovery room were unchanged. A repeat analysis of arterial blood gas levels showed a pH of 7.38, P_{CO_2} of 45 torr, and P_{O_2} of 71 torr. He was then admitted to the hospital for close observation. He received no treatment except for oxygen. Twenty-four hours after the onset of pulmonary edema, his abnormal chest x-ray findings resolved completely, and the P_{aO_2} on room air increased to 93 torr. His subsequent course was uneventful, and he was discharged 30 hours after the acute episode.

Discussion

Review of the pertinent literature reveals that laryngospasm has been the most common cause of upper airway obstruction leading to NPPE in anesthesia in adults.[1,6,9] However in the pediatric population, epiglottitis, croup, and foreign bodies account for more than 80% of reported cases.[1] The upper airway obstruction in this case, however, was induced by biting on the endotracheal tube. DeSoto and Johnston[10] have reported a similar case in which biting on an endotracheal tube for only 20 seconds induced NPPE in a child. They focused on the necessity of a bite-block during anesthesia. We did not use an oral airway as a bite-block because they have been shown to cause sore throat postoperatively.[11]

NPPE tends to occur in young healthy persons rather than in the elderly. The pathogenesis is multifactorial and believed to be primarily related to the development of markedly negative intrapleural pressures augmented by hypoxia and a hyperadrenergic state. Blood return to the right heart is abruptly increased secondary to increased venous return and a redistribution of systemic volume to the central circulation. Left heart output is decreased due to increased afterload, acidosis, and hypoxia. The resultant increase in pulmonary blood volume and hydrostatic pressures along with negative interstitial pressures and increased capillary permeability facilitate the development of pulmonary edema. The pathophysiologic mechanism of NPPE, which is identified by rapid onset and marked intrapleural negative pressure, may be similar to that of re-expansion pulmonary edema secondary to rapid drainage of pneumothorax or intrathoracic fluids.[1]

In this case, the patient was young and muscular. Transpulmonary pressure gradients must have increased tremendously because of the patient's vigorous inspiratory efforts against the airway obstruction. The pink frothy sputum appearing in the endo-tracheal tube soon after the relief of obstruction implies that the NPPE developed rapidly. Therefore, the marked decrease in oxygen saturation might have been the result not only of hypoventilation caused by tube obstruction but also of impairment in alveolar gas exchange caused by developing pulmonary edema.

Finally, an oropharyngeal airway was inserted to relieve the patient's biting on the endotracheal tube. A small dose of intravenous succinylcholine should be considered for that purpose.

Pulmonary edema in this patient resolved within 24 hours without aggressive treatment (such as continuous positive airway pressure or diuretics). Willms and Shure[6] and Lorch and Sahn[9] found that most NPPE cases resolve spontaneously and do not require aggressive therapies. The good health of many of these reported patients may explain this rapid improvement, and possibly why severe destruction of capillary-interstitial-alveolar microstructures rarely occurs. Nonetheless, hypoxemia may persist despite oxygen therapy after development of NPPE, and mechanical ventilation with positive airway pressure may be required.[1]

This patient also received the opioid antagonist naloxone prior to extubation. Adverse side effects have been reported following its administration, in-

cluding hypertension,[12] pulmonary edema,[13,14] ventricular arrhythmias,[15] and cardiac arrest.[16] Reported cases of naloxone-induced pulmonary edema have involved higher total doses (0.3-0.4 mg)[13,14] than administered in this case. We believe that the small dose of naloxone and the identification of pulmonary edema simultaneously with its administration exclude naloxone as a causative agent in this case.

Although the majority of cases of NPPE develop shortly after the onset of airway obstruction, delayed onset of NPPE has been reported.[1,6,7] Delayed onset indicates that careful observation is required for several hours after an episode of upper airway obstruction from any cause.

In this case, we identified a simple mishap, a bite on an endotracheal tube, as a possible cause of NPPE in a healthy adolescent patient. We advocate routine use of a bite-block during anesthesia to avoid this complication.

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Patient-Ventilator Interaction: Rational Strategies for Acute Ventilatory Management

John J Marini MD

Introduction

The term patient-ventilator interaction applies to a great number and variety of events that occur during machine-assisted breathing. Because this topic is too extensive to attempt any comprehensive review, I have chosen to focus on two types of interaction. The first addresses the ways in which the setup of the machine influences the work, comfort, and synchrony of patient and ventilator-assisted breathing cycles. The ventilated patient with severe airflow obstruction serves to exemplify these points. The second type of patient-ventilator interaction concerns the potential for ventilator-induced tissue injury. For this, I will concentrate on the ventilatory management of the adult respiratory distress syndrome.

Work, Comfort, and Synchrony

Dynamic hyperinflation is a key feature of the ventilatory process in patients with severe obstructive lung disease.[1-4] Such patients must breathe from a high lung volume in order to dilate airways and generate sufficient expiratory recoil to expel the tidal volume in the time allowed between breaths.[1,3,4] Gas trapping flattens the diaphragm and disadvantages the respiratory musculature, reducing the patient's ability to accomplish the ventilatory workload.[2] Moreover, dynamic hyperinflation is invariably associated with what has come to be known as auto- or intrinsic PEEP. Auto-PEEP is the flow-driving pressure difference between the alveolus and the external circuit that develops at end-exhalation.[3,4] It is this pressure gradient that drives flow through severely narrowed airways to the very end of exhalation—until interrupted by the next inflation cycle. When Pepe and I first reported this phenomenon, we entitled our paper "*Occult* Positive End-Expiratory Pressure in Ventilated Patients with Air Flow Obstruction—the Auto-PEEP Effect."[3] Auto-PEEP is occult because it does not become evident to the respiratory care practitioner until expiratory flow is deliberately stopped or the expiratory flow tracing is carefully examined. There are many ways to estimate auto-PEEP, and the expiratory port occlusion maneuver that we originally used is just one of these. Most other techniques involve measuring the applied airway or muscular pressures required to terminate the end-expiratory flow that auto-PEEP generates.[5-8]

In the passively ventilated patient, the determinants of auto-PEEP are those that are influenced by the impedance to deflation and those that are determined by the level of ventilation and the cycling rhythm. Increases in the expiratory time constant (the product of resistance and compliance), increases of the inspiratory time fraction (leaving less time for exhalation), and increases in minute ventilation raise alveolar pressure at end-exhalation. In practice, reducing minute ventilation is perhaps the most potent—and most commonly overlooked—measure that can be taken to reduce gas trapping.

In our original paper, we illustrated the potent hemodynamic consequences of high level auto-PEEP in a patient with compliant lungs.[3] When the patient was connected to the ventilator, the cardiac

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output and systemic blood pressure remained at shock levels, despite a high pulmonary artery wedge pressure and the use of inotropes (Fig. 1). When the ventilator was temporarily disconnected to allow airway suctioning, the blood pressure and cardiac output increased markedly, even as the measured wedge pressure fell. These observations are consistent with the behavior expected when elevated alveolar pressure increases intrapleural and pulmonary vascular pressures. Auto-PEEP raises the wedge pressure but reduces its transmural pressure equivalent, and, consequently, diminishes preload. Although dynamic hyperinflation also causes a volume-associated increase of right ventricular afterload, this is usually of less significance.

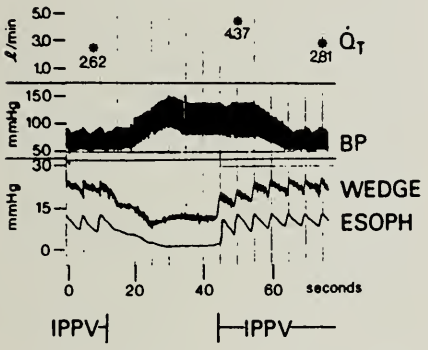

Fig. 1. Hemodynamic effect of dynamic hyperinflation. Hypotension, diminished cardiac output ($\dot{Q}_T$), and elevated wedge pressure observed during mechanical ventilation (IPPV) are reversed during a 30-second period of apnea. Marked elevation of wedge pressure and depression of venous return are caused by increased intrapleural pressure (ESOPH). (Reprinted from Reference 3, with permission.)

It is very important for respiratory care practitioners to understand that this same phenomenon can happen during cardiopulmonary resuscitation, as well. When the patient is ventilated too rapidly—manually or by a mechanical ventilator—blood pressure can fall to undetectable levels, even in the presence of a coordinated electrocardiogram.[9] This, of course, is viewed as "electromechanical dissociation" or more accurately perhaps "pulseless electrical activity," which ordinarily is a very grave prognostic sign. Resuscitation efforts may even be terminated prematurely, only to have the team begin again when an effective pulse re-emerges as positive pressure ventilation ceases. Blood pressure should be remeasured after an extended period of apnea whenever electromechanical dissociation or severe hypotension are encountered in this setting. For patients with severe air trapping, more than 20 seconds may be required to effect full reversal of dynamic hyperinflation.[10]

The hemodynamic consequences of dynamic hyperinflation depend not only on underlying cardiac function and the magnitude of auto-PEEP, but also on the status of vascular filling, the afterload to the right ventricle, the integrity of compensatory vasomotor reflexes, and the existence of spontaneous breathing efforts. Of these factors, the last is among the most important. During spontaneous breathing, the *average* intrapleural pressure experienced by the patient with severe airflow obstruction is often *lower* than normal, despite hyperinflation. This apparent paradox was described more than 15 years ago in children with asthma by Stalcup and Mellins,[11] and more recently has been observed in adults in both laboratory[12] and clinical settings.[13] During spontaneous breathing, the vigorous inspiratory and expiratory pumping actions develop pressure gradients that effectively propel blood from the periphery to the thorax. Venous return may fall precipitously when the ventilator develops positive pressure in the thorax while the venous bed—vasodilated by sedatives, unsupported by relaxed muscles, and often underfilled by dehydration—cannot compensate.

Conversely, the institution of mechanical ventilation may benefit patients with left ventricular dysfunction or ischemia. Reducing the work of breathing alleviates excessive demands for cardiac output. In addition, positive intrathoracic pressure can reduce the increased left ventricular afterload and high transvascular pressures associated with vigorous spontaneous breathing. When forceful respiratory efforts are silenced, pleural pressure rises, reducing the tendency for pulmonary congestion. Thus, whether initiating mechanical ventilation in the patient with severe airflow obstruction has a beneficial or detrimental hemodynamic effect depends on the underlying status of the heart and vascular system.

Although auto-PEEP may not threaten cardiac output during spontaneous breathing, it becomes a potential cause of muscle fatigue. The mechanical work of breathing can be quantified as the area enclosed by the pressure applied across the respiratory system plotted against the volume that it moves.[14] In breathing against elevated resistance, the patient with severe airflow obstruction has increased inspiratory work to accomplish. Moreover, the hyperinflated lung rests higher on the pressure-volume curve of the respiratory system, thereby reducing compliance and increasing the elastic work of tidal inspiration, as well. Of crucial significance, auto-PEEP (an expiratory pressure) must be counterbalanced for inspiration to begin at all. Auto-PEEP therefore acts as an inspiratory threshold load and accounts for a substantial amount of inspiratory work. How important is the work associated with auto-PEEP in comparison to the other components of the work equation? The respiratory muscles, compromised by hyperinflation, are functionally weak and, therefore, may have the capacity to generate a maximum inspiratory pressure of only 30-50 cm H_2O. The maximum *tidal* pressure that can be sustained without fatigue may be only half as great, or 15-25 cm H_2O per breath.[15] Auto-PEEP levels of 5-15 cm H_2O, values well within the range observed clinically, will severely load this compromised system.

During machine-assisted breathing, auto-PEEP represents a positive pressure that must be overcome before the ventilator can be activated. By definition, auto-PEEP is the positive difference between alveolar and circuit pressures just before inspiration begins. To initiate inflation, pressure in the central airway must fall to a value 1-2 cm H_2O less than the set PEEP level. The patient experiencing dynamic hyperinflation must first counterbalance the positive auto- (or intrinsic) PEEP before the central airway pressure can be reduced by this amount.[5,16] *Effective* triggering sensitivity, therefore, is markedly depressed.

The difficulty that auto-PEEP causes in triggering the ventilator often manifests as intermittent failure of patient efforts to initiate an assisted cycle. At the bedside, we observe the subject's attempting to trigger the ventilator more frequently than the observed number of machine-aided breaths—with only the most vigorous efforts succeeding. Obviously, this can increase the subject's work of breathing and dyspnea, reduce overall ventilation, or unintentionally convert *synchronized* to *unsynchronized* intermittent mandatory ventilation (IMV). To avert these detrimental effects the practitioner should first attempt to reduce auto-PEEP by improving the expiratory time constant, providing more expiratory time, or reducing the minute ventilation requirement.[17] As a general rule, manipulating the frequency-tidal volume combination yields relatively little benefit unless minute volume is reduced simultaneously.

Another approach to alleviating the detrimental effects of auto-PEEP on breathing effort is to counterbalance end-expiratory alveolar pressure with PEEP deliberately applied at the airway opening[5-7] (Fig. 2). Until a few years ago, this approach might have been strongly discouraged because it was generally believed that conventional PEEP would cause further hyperinflation. Whether PEEP helps or exacerbates the problem is largely determined by the nature of the conditions that give rise to auto-PEEP.[16]

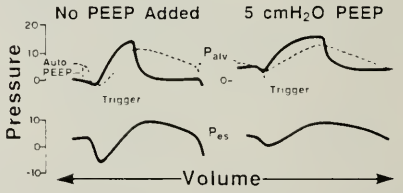

Fig. 2. Effect on triggering sensitivity of adding PEEP to auto-PEEP during an assisted machine cycle. To initiate the positive pressure cycle, the patient must reverse auto-PEEP, as well as overcome the set trigger sensitivity (left panel). Adding PEEP downstream of the site of flow limitation narrows the difference between alveolar (Palv) and airway pressures, improving effective triggering sensitivity and breathing effort. (Reprinted from Reference 4, with permission.)

To illustrate, consider two patients, each with 10 cm H_2O auto-PEEP, but differing with regard to the existence of flow limitation during tidal expiration. The first patient has a relatively fixed (structural) resistance in the airways or external expiratory circuit, a high ventilatory requirement, and no limitation to expiratory airflow. Such conditions often pertain to the adult respiratory distress syndrome (ARDS). Assuming that tidal volume and

minute ventilation do not change, adding PEEP predictably increases lung volume. Here, although PEEP may recruit lung volume or prove advantageous for other reasons, it will fail to reduce the work of breathing associated with auto-PEEP.

On the other hand, the patient with dynamic (functional) airway compression and expiratory flow limitation essentially performs the equivalent of forced spirometry with each tidal breath because he or she breathes on the expiratory flow-volume envelope.[16,18] The main determinant of the rate of lung emptying is the difference between the alveolar pressure and the critical pressure that limits airflow. Although somewhat less than end-expiratory alveolar pressure, the critical pressure at the point of airway compression exceeds that in the external circuit.[18] Adding pressure downstream from this site fails to influence alveolar pressure until the added pressure exceeds the critical value, much like the classical waterfall analogy used to explain pulmonary vascular "zone 2" conditions.[19,20] The addition of PEEP less than the critical pressure does not change the alveolar pressure, but *does* narrow the difference between alveolar and central airway pressures, thereby improving effective trigger sensitivity. As a general rule, critical pressure averages about 80% of the baseline auto-PEEP value. Once the critical pressure is counterbalanced, further additions of PEEP cause parallel increases of alveolar pressure.[5,18]

At the bedside, the clinician can gauge the appropriate value of added PEEP by its effect on peak inflation and end-inspiratory plateau pressures.[5,16] PEEP can be added safely until further increases cause peak inflation pressures to rise in step. For directly analogous reasons, adding CPAP to a spontaneously breathing subject with dynamic hyperinflation can make the natural breath substantially easier to draw.[6,21]

Patient-Ventilator Synchrony and Breathing Workload

For many years now, synchronized intermittent mandatory ventilation (SIMV) and pressure support ventilation (PSV) have been the primary modes of partial ventilatory assistance.[22,23] Although both are designed to augment patient efforts, SIMV differs from PSV with regard to the airway pressure profile, the proportion of breaths supported by the machine, and the primary off-cycling criterion. When SIMV is in use, the machine delivers a clinician-selected number of breaths per minute with a specified volume and a stereotyped flow profile. The patient performs all work associated with non-assisted breaths. This strategy is unlike PSV, a mode in which every cycle is partially assisted to the same pressure level.[23,24] PSV is designed to allow the patient to exert more control over the flow profile and duration. The proportion of the ventilatory workload off-loaded to the machine depends on the pressure setting and on the patient's underlying requirement, strength, breathing pattern, and ventilatory drive.

A number of years ago, my colleagues and I began a series of studies that attempted to define the determinants and quantify the work associated with the machine-aided breaths of assist/control (AMV) and SIMV.[25-27] During the machine assisted, volume-cycled breaths of either mode, the unstated goal is for the patient simply to trigger the machine, while the ventilator assumes the great majority of the inflation workload. In practice, however, the subject seldom stops the breathing effort abruptly once it has begun, so that the ventilator and patient act together to accomplish the work associated with the breath. For a given machine-aided cycle, the more vigorously the subject pulls, the less effectively is work off-loaded to the machine.[25]

As our experiments demonstrated, the main determinants of effort during a flow-controlled, volume-cycled breath are those that relate to ventilatory drive: minute ventilation, trigger sensitivity, and flow delivery rate.[25,26] Although other comfort issues (including the size of the tidal volume and the magnitude of applied PEEP) may also affect drive and patient effort, their impact on patient effort has not been carefully studied.

SIMV does not always relieve the patient's breathing workload as intended. This was made evident to us when we compared the breathing efforts of spontaneous and machine-assisted cycles at a fixed level of chemical drive (P_{aCO_2} and pH). We made such a comparison in a sample of 15 ventilator-dependent patients in whom we transiently withdrew ventilatory support in stages ranging from AMV (full support) to spontaneous breathing.[27] At each intermediate level (80, 60, 40, or

20% of the machine frequency of AMV), the inspiratory pressure-time products for machine-assisted cycles proved little different from those of spontaneous cycles. The machine certainly helped with the patient's overall rate of energy expenditure because the work per breath, breathing frequency, and ventilatory drive each increased as support was withdrawn. On a breath-to-breath basis, however, these dyspneic patients were indifferent to the loading status of the respiratory muscles and did not significantly modify their effort (pressure-time product) once the inspiratory cycle had been initiated. The stressed patient did not appear to adapt to the relief of muscular afterload that the machine could potentially provide. As judged by esophageal rather than airway pressure, one effort appeared very much like the next—machine aided or not. (This has recently been confirmed by quantitative electromyography of the diaphragm.[28]) In this limited sense, at least, SIMV and PSV are perhaps not that different after all.

Not only does the dyspneic patient fail to take full advantage of the power assist that the machine could potentially provide, but he or she also adapts poorly to the imposed cycling pattern. Inability of the patient to conform to the rigidly imposed cycle length dictated by the volume-cycled mode of ventilation is reflected in the timing dysynchrony so often seen in the setting of poorly adjusted assist-control ventilation. When the flow setting is maladjusted to the patient's intrinsic need, the muscles can continue to pull after the machine has finished pushing. Conversely, the patient may cycle into exhalation before the ventilator has completed its inspiratory cycle. At the bedside, deformation of the airway pressure waveform and variations in peak cycling pressure are important to notice when one is making adjustments aimed at synchronizing the patient's duty cycle and flow demands with those imposed by the ventilator.[29]

Pressure support ventilation has been generally regarded as superior to flow-controlled, volume cycled ventilation in coordinating with the patient's cycling rhythm, and for many clinical settings, its superiority is clearly evident.[23,24] After the PSV-supported cycle is initiated by the patient, pressure rises toward the set plateau level at a rate determined by the type of machine in use. Some of the newer ventilators allow adjustment of the rate of pressure buildup, and such adjustability may be useful in adapting the machine's output to variations in patient requirements. Slower rates are appropriate for quieter breaths, whereas rapid rates are desirable for vigorous breathing efforts.

Pressure support is designed to allow the subject to control the ventilatory cycle, not only in timing its onset, but also in regulating the velocity of gas entering the chest and the length and depth of the tidal breath. During the pressure-assisted inspiration, flow decays from a peak value achieved early in the cycle to some criterion value needed for the ventilator to initiate the expiratory phase.[23] On most ventilators, this flow-governed 'off-switch' is either a specific inspiratory flowrate or a fixed percentage of the observed peak flow value. Many clinicians believe that the use of pressure support provides virtually unlimited flow potential, but this is not really so—especially at low pressure settings. The maximal flowrate that PSV can achieve is determined by the interaction of several factors: the magnitude of the applied pressure (P_{set}), the abruptness of the pressure rise, the inspiratory resistance (R_I), and the vigor of patient effort. With minimal patient effort and a perfectly 'square' wave of applied pressure, the maximal inspiratory flowrate is P_{set}/R_I in L/s. For example, 10 cm H_2O PSV acting through a resistance of 10 cm $H_2O \cdot L^{-1} \cdot s^{-1}$ would yield a peak inspiratory flow of 1 L/s. Faster flows are achievable only at the direct cost of more patient effort or an increase of P_{set}.

Many patients with severe airflow obstruction are *not* ideally suited to pressure support as the sole power source providing assistance, particularly when breathing pattern, inspiratory impedance, and auto-PEEP change abruptly. Such patients often have variable minute ventilation and power requirements, and this variability makes it difficult for the practitioner to select a single pressure value that assures an appropriate support under changing conditions. For example, if the patient is breathing rapidly, a high level of pressure support is generally required to overcome the resistance to airflow; but, just as soon as breathing frequency slows or resistance diminishes, tidal volume can increase dramatically as the pressure in the compliant lungs rises toward equilibration with high airway pressure. On the other hand, the machine's percentage of the ventilatory workload and the size of the tidal vol-

ume can both fall dramatically when a debilitated patient increases breathing frequency. Certain newer forms of ventilation regulate the applied pressure or supply supplemental flow as needed to achieve a targeted tidal volume.[30] Although these may well be somewhat closer to offering the ideal strategy, in my view they are still imperfect.

A more subtle problem often occurs with the off-switch to airway pressurization. Under passive conditions, the flow waveform of patients with severe airflow obstruction tends to assume a squared configuration. Airflow may simply decelerate too slowly under passive conditions to achieve the off-switch criterion value in time for exhalation to begin synchronously with the patient's intrinsic rhythm (Fig. 3). Exhalation must then be initiated by expiratory muscle action. Apart from adding to the total work of breathing, increased expiratory pressure might even increase the severity of obstruction itself by worsening dynamic airway compression and producing 'negative' effort dependence.[31]

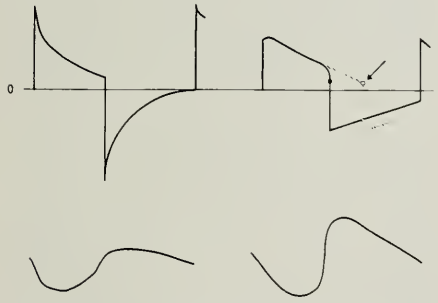

Fig. 3. Off-switch triggering during pressure-controlled ventilation for normal and restricted patients (left) and for a severely obstructed patient (right). 'Squared' flow profile of inspiration during airflow obstruction may require the patient to use expiratory effort to halt flow sufficiently to satisfy the ventilator's end-inspiratory cycling criterion. Passive exhalation (dashed line) would require excessive inspiratory time to achieve 25% of peak flow (arrow).

As with any pressure-limited mode, the ventilatory effectiveness of PSV varies as impedance changes. Bronchospasm, airway secretions, and auto-PEEP interfere with its effectiveness; therefore, a given support level that was initially effective early in a weaning trial may prove insufficient to meet the needs of the patient some minutes after that trial begins.

One of the most exciting concepts in the attempt to synchronize machine output with patient requirements has been proposed by Younes and his colleagues at the University of Manitoba in Winnipeg.[32,33] Termed proportional-assist ventilation (PAV), this mode aims to match machine output with patient effort, thereby allowing the machine to adapt fluidly to the patient's needs for flow and power. With appropriately adjusted PAV, the patient determines the underlying cycling rhythm, while the ventilator acts as a powerful accessory muscle. Proportional assist is on the near horizon but not currently available for clinical application.

Ventilator-Induced Lung Injury

A second major category of adverse patient-ventilator interactions concerns the potential for mechanical ventilation to injure the lung. This discussion is perhaps most pertinent to the patient with acute lung injury (ARDS). For many years, it was assumed that lung damage in ARDS was diffusely distributed. It is now known, however, that severely damaged and normally compliant areas co-exist in different regions.[34,35] Conceptually, the injured lung might be considered to be composed of three separate compartments: normal, densely consolidated, and potentially recruitable. Although the frontal chest x-ray suggests *diffuse* lung damage, the mechanics of ARDS are likely to be quite heterogeneous. Early in the disease process the aerated compartment of the lung is *small*, not inherently stiff.

Gattinoni,[35] Maunder,[36] and their respective colleagues showed this heterogeneity nicely with transverse computed tomographic sections of the thorax. Early in the course of ARDS, dependent regions appear densely consolidated, whereas anterior regions tend to remain relatively lucent. When high pressure is applied to the airway opening, some volume is recruited from the dependent areas, but the nondependent regions become overinflated.

Overdistention produced by the application of large tidal volumes and pressures may rapidly disrupt the integrity of the alveolar capillary membrane, even without causing overt rupture. In seminal work completed almost 20 years ago, Webb

and Tierney[37] exposed three sets of normal rats to different patterns of mechanical ventilation for a period of a single hour.[37] Normal ventilating pressures of 14 cm H_2O peak airway pressure resulted in no damage, as expected. However, a peak inflation pressure of 45 cm H_2O pressure without PEEP caused grossly hemorrhagic pulmonary edema. It is noteworthy that adding 10 cm H_2O PEEP limited the damage resulting from the same high peak inflation pressure (45 cm H_2O) to interstitial edema. The exact reason for PEEP's protective effect is not known. Perhaps PEEP kept areas from collapsing that were injured by high inflation pressure. Alternatively, PEEP may have averted damage resulting from large tidal *excursions* of pressure or volume. The important point of this work and corroborative findings from many other animal studies[38-41] is that normal lung tissue can be damaged by high pressures, and perhaps by large tidal volumes. Moreover, failure to maintain a certain minimum volume may have serious consequences once injury has been initiated.[37,42] If the acutely injured lung contains many 'normal' areas that perform the majority of gas exchange, as radiographic, densitometric, and physiologic data imply, these regions may be at risk for the same kind of damage.

Following the clues yielded by many studies of pressure-induced lung injury, the essential principles of a 'protective' strategy for ventilating the patient with ARDS can be summarized as follows: First, prevent alveolar overdistention. Second, maintain some minimum end-expiratory lung volume in the early phase of the disease process, generally with total PEEP (PEEP + auto-PEEP) values of 10-15 cm H_2O. Third, avoid using large tidal volumes. Fourth, use the minimum necessary mean airway pressure required for adequate arterial oxygenation. High mean airway pressures may depress cardiac output, impair oxygen delivery, or extend pre-existing barotrauma.[43,44]

A number of approaches have been proposed with these ventilatory objectives in mind. Pressure-controlled ventilation,[45] inverse ratio ventilation,[46] and the variants of airway pressure-release ventilation[47,48] have all been suggested to be potentially useful. In each of these, a fixed airway pressure is applied and released as pressure at the alveolar level builds and decays in exponential fashion. Rigid control can, therefore, be exercised on the maximum pressure to which the alveoli are exposed. Total transalveolar pressure, the critical value for the generation of tissue injury, is the difference between alveolar and intrapleural pressures. During passive inflation, this pressure is predictably less than the set airway pressure; during vigorous breathing efforts, it may be substantially more.

With pressure control, the clinician can adjust only four ventilatory settings: (1) the applied level of positive end-expiratory pressure, (2) the maximum pressure to which the airway is subjected, (3) the duty cycle length t_i/t_{tot} (ie, the inspiratory time as a per cent of respiratory cycle time), and (4) the breathing frequency.[17,45] Because alveolar pressure builds and decays exponentially, some of the ventilating pressure is dissipated against resistance if insufficient time is allowed for the airway and alveolar pressures to equilibrate, either at end-inspiration or end-expiration. Pressure control allows the clinician to set the maximal and minimal pressures to which the alveolus can be exposed—two major objectives in the ventilation of patients with ARDS. What suffers, however, is the clinician's ability to control ventilation, which is a complex function of the patient's impedance to breathing and the setting selections.[17]

When the inspiratory duty cycle is varied over a wide range with set values for pressure and frequency, an inverted U-shaped curve is described for the relationship between minute ventilation and t_i/t_{tot}.[49] However, the configuration of this curve differs with the mechanical properties of the respiratory system (Fig. 4). With severe restriction, alveolar and airway pressures equilibrate rapidly during each cycle for all duty cycles except those that are extremely short or long, and there is no single optimal value for t_i/t_{tot}. For severe airflow obstruction, however, there is a sharply defined optimum.

What happens when t_i/t_{tot} and applied pressure remain fixed and *frequency* is varied? Again, it depends on whether you are dealing with obstruction or restriction (Fig. 5). As frequency rises, tidal volume progressively declines because the time available becomes too brief to allow equilibration between alveolar and airway pressures. The rate at which tidal volume falls off with increasing frequency is more precipitous for obstructive than for restrictive disease. With P_{aet} fixed, there is a certain minute ventilation that cannot be exceeded, no matter how t_i/t_{tot} and frequency are manipulated.[17,49] At

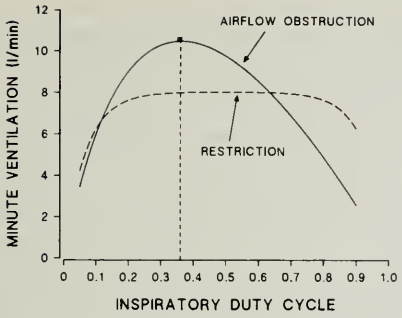

Fig. 4. Relationship of minute ventilation to inspiratory duty-cycle length for pressure-preset ventilation in patients with severe airflow obstruction and restriction (computer simulation). Note distinct optimum duty-cycle length for the obstructive case. (Reprinted from Reference 49, with permission.)

a specific t_i/t_{tot}, the upper limit for ventilation level is a function only of applied ventilating pressure and the resistance of the airway. Variations in compliance influence the rate at which this upper limit is approached with increasing frequency, but do not influence the value itself (Fig. 5).

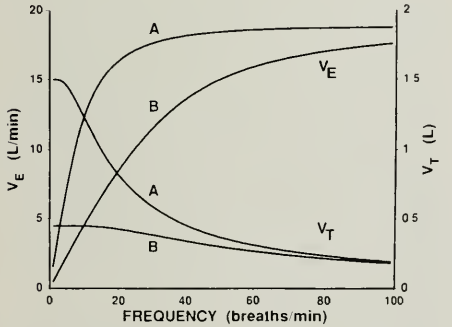

Fig. 5. Relationship of minute ventilation and tidal volume to breathing frequency during pressure-preset ventilation (computer simulation). Conditions A and B only differ with respect to the compliance of the respiratory system. The approach to the minute ventilation plateau and the fall in tidal volume with increasing frequency are sharper for the normal compliance condition (A) than for the low compliance condition (B). (Reprinted from Reference 49, with permission.)

Not only is the maximum minute ventilation fixed, but as frequency and minute ventilation increase, so does the wasted portion of each tidal breath—the physiologic dead space fraction, V_D/V_T. This deterioration occurs because the dead space fraction rises as tidal volume falls. In normal dogs, a species with a relatively large anatomical dead space volume, V_D/V_T rises rather sharply at all frequencies greater than 20-25/min.[50]

In experiments we have conducted in St Paul, minute ventilation approached the plateau predicted by our mathematical model of pressure preset ventilation,[17,50] and P_{aCO_2} rose inexorably with progressive increases in cycling frequency. Such data are rather disturbing because they imply that if we constrain pressure and/or tidal volume excursions for lung protection (as the experimental data imply), we are committed to hypoventilation and CO_2 retention. Permissive hypercapnia may be part and parcel of a lung protection strategy. The question then arises, Is hypercapnia always detrimental? The appropriate answer probably lies with the the type and severity of illness and the rapidity with which CO_2 is retained.

Apart from stimulating respiratory drive, the consequences of CO_2 retention relate to intracellular pH changes and include muscle weakness, intracranial hypertension, and dysfunction of the central nervous and cardiovascular systems.[51] Chronic problems with CO_2 retention, however, may not be significant. The wisdom of using a pressure limiting strategy that incorporates permissive hypercapnia has been suggested by at least two retrospective studies. The first was published by Darioli and Perret in 1984 for status asthma,[52] the other by Hickling in 1990 for ARDS.[53] (The rationale for permissive hypercapnia has been reviewed by Kacmarek and Hickling in this journal.[54])

Unfortunately, many patients ventilated with permissive hypercapnia require deep sedation and paralysis, at least during the period of accommodation to the higher P_{aCO_2} and in cases where inverse ratio ventilation is required. Institution of paralysis can restore balance to the oxygen-delivery-to-consumption relationship, improve P_{aCO_2}, and reduce the cardiac workload.[55] However, muscle relaxants (paralytic agents) must be discontinued as soon as feasible to do so. Current evidence strongly

suggests that prolonged blockade of the neuromuscular junction can cause residual weakness requiring weeks to months for recovery.[56-59] Episodic reversal of paralysis or close monitoring of the depth of paralysis to allow neuromuscular transmission may be required to prevent such complications.

At the current time, my own approach to avoiding pressure-related tissue injury is to first assess the need for muscle relaxants (Fig. 6). If there are no clear indications (generally in those patients with moderate disease), I typically use SIMV with pressure support and CPAP. Others may choose strategies such as airway pressure release[47] or its variant, intermittent pressure release ventilation (bi-level CPAP).[48] If the situation deteriorates, deep sedation and/or paralysis may be needed, and then my vote is for a pressure-targeted strategy that includes extended ratio ventilation and permissive hypercapnia as integral components.[46] I deliberately use the adjective *extended*, rather than *inverse*; as my colleagues and I have previously emphasized,[46,60] inverse ratio ventilation may be a logical result of this approach, but should not be either the routine starting point or the technique used when all others have failed. Often, the t_i/t_{tot} does not need to be extended to the point of ratio inversion to achieve good results.

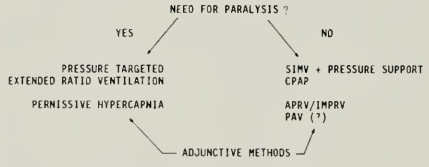

Fig. 6. Possible modes for pressure-limited ventilation.

In some centers, experimentation is underway with adjunctive measures that aim to eliminate carbon dioxide without using high ventilating pressures or large lung excursions. Such aggressive approaches include extracorporeal CO_2 removal ($ECCO_2R$)[61] and intracaval gas exchange achieved via a femoral vein catheter composed of many gas-permeable hollow fibers (IVOX).[62]

Tracheal gas insufflation (TGI), a third, less invasive adjunct to pressure-limited conventional ventilation, appears to hold considerable promise. We and others have had some initial clinical experience with TGI as a method to simultaneously reduce the minute ventilation requirement without violating the principles geared to avoid adverse patient-ventilator interactions.[63,64] Our team was inspired to pursue this line of investigation by our own modeling experiments with pressure-preset ventilation[17,50,65] and by the intriguing experiments of other workers using similar techniques in other settings. Here the work of such investigators as Slutsky,[66,67] Kolobow,[68] Hurewitz,[69] Bergofsky,[70] Sznajder,[71] and Stresemann[72,73] and their colleagues have been influential. In the majority of their studies, the fresh gas injected into the trachea cleared some of the dead space in the proximal airway during exhalation, reducing the minute ventilation or ventilatory pressures required to accomplish a given P_{aCO_2}.

In a series of experiments in dogs[50,74,75] and patients,[63] we used a catheter placed alongside or through an endotracheal tube to inject fresh gas near the carina, either continuously or phasically in exhalation. Tracheal injection of fresh gas works in conjunction with pressure-preset ventilation to accomplish effective ventilation without exposing the lung to excessive tidal pressure or volume excursions.

During the inspiratory phase of continuous TGI, the catheter acts as a miniventilator whose hypothetical Y-point (ie, the point of separation of inhaled and exhaled gas streams) is positioned very near the carina, bypassing the proximal airway.[68,75] During the expiratory phase, the anatomic dead space cephalad to the injection site is washed out by the fresh gas flow. As the catheter helps to build pressure to the targeted level, the pressure-controlled ventilator throttles down its flow output. A similar effect can be achieved by manually reducing the ventilator's contribution to the total inspired tidal volume during flow-controlled, volume-cycled ventilation.[75]

Our first experiments in normal dogs demonstrated that modest catheter flows (2-10 L/min) caused P_{aCO_2} to fall dramatically, even as total tidal volume and ventilation pressures remained constant.[50] Variations of catheter shape and orifice location did not produce dramatic differences and the exact position of the catheter tip was relatively inconsequential to the observed result, provided that the orifice was reasonably close to the carina. This

is quite unlike continuous flow ventilation, a mode without phasic variations of ventilating pressure.[67,76]

In other work, we demonstrated that similar but more modest reductions of P_{aCO_2} could be accomplished by tracheal gas insufflation in human subjects receiving mechanical ventilation.[63] In a sample of critically ill patients with diverse lung pathology, TGI substantially improved the alveolar ventilation accomplished at the same total inspired V_T. Although such techniques have not yet been fully developed or tested, we are optimistic that they may eventually help to limit carbon dioxide retention during pressure-limiting strategies of ventilation. TGI might also help to accomplish effective ventilation in the weaning process, especially for patients whose primary problem is neuromuscular weakness. At this time (1992), however, these techniques remain experimental.

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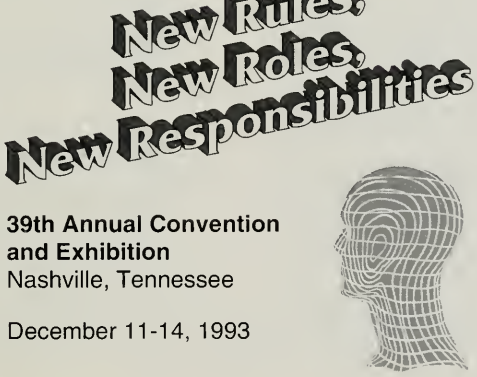

39th Annual Convention and Exhibition
Nashville, Tennessee

December 11-14, 1993

Update on Clinical Practice Guidelines

Five new AARC Clinical Practice Guidelines join the 13 previously published. The new Guidelines are

Directed Cough

Endotracheal Suctioning of Mechanically Ventilated Adults and Children with Artificial Airways

In-Vitro pH and Blood Gas Analysis and Hemoximetry

Single-Breath Carbon Monoxide Diffusing Capacity

Use of Positive Airway Pressure Adjuncts to Bronchial Hygiene Therapy

The first five Guidelines appeared in the December 1991 issue of RESPIRATORY CARE. Eight more were published in the August 1992 issue. Those Guidelines and their citations are shown below.

Incentive Spirometry [Respir Care 1991;36(12):1402-1405]

Pulse Oximetry [Respir Care 1991;36 (12):1406-1409]

Oxygen Therapy in the Acute Care Hospital [Respir Care 1991;36(12):1410-1413]

Spirometry [Respir Care 1991;36(12):1414-1417]

Postural Drainage Therapy [Respir Care 1991;36 (12):1418-1426]

Patient-Ventilator System Checks [Respir Care 1992;37(8):882-886]

Humidification during Mechanical Ventilation [Respir Care 1992;37(8):887-890]

Selection of Aerosol Delivery Device to the Lower Airways [Respir Care 1992;37(8):891-897]

Nasotracheal Suctioning [Respir Care 1992;37(8):898-901]

Bronchial Provocation [Respir Care 1992:37(8):902-906]

Exercise Testing for Evaluation of Hypoxemia and/or Desaturation [Respir Care 1992;37(8):907-912]

Sampling for Arterial Blood Gas Analysis [Respir Care 1992;37(8):913-917]

Oxygen Therapy in the Home or Extended Care Facility [Respir Care 1992;37(8):918-922]

Additional Guidelines are scheduled for publication in the August 1993 issue of RESPIRATORY CARE.

AARC Clinical Practice Guideline

Directed Cough

DC 1.0 PROCEDURE:

Directed cough (DC) to clear or mobilize secretions is a component of bronchial hygiene therapy when spontaneous cough is inadequate. Directed Cough is a deliberate maneuver that is taught, supervised, and monitored. Forced expiratory technique (FET, or huff cough) and manually assisted cough are examples of directed cough.

DC 2.0 DEFINITION/DESCRIPTION:

Directed cough seeks to mimic the attributes of an effective spontaneous cough (or series of coughs), to help to provide voluntary control over reflex, and to compensate for physical limitations (eg, by increasing glottic control, inspiratory and expiratory muscle strength, coordination, and airway stability).

A spontaneous effective cough is a reflex mechanism utilizing maximum forced exhalation to clear irritants or secretions from the airway. The forced exhalation is preceded by a maximal inspiratory effort followed by closure of the glottis. Contraction of expiratory muscles produces increased intrathoracic pressures against the closed glottis, which culminates in an explosive release of gas at high velocity as the glottis opens.

Forced Expiratory Technique (FET), also known as "huff coughing," consists of one or two huffs (forced expirations) from mid-to-low lung volumes with the glottis open,[1] followed by a period of relaxed, controlled diaphragmatic breathing. The process is repeated until maximal bronchial clearance is obtained, and can be reinforced by self-compression of the chest wall using a brisk adduction movement of the upper arms.[2]

Manually assisted cough is the external application of mechanical pressure to the epigastric region or thoracic cage coordinated with forced exhalation.

DC 3.0 SETTING:

3.1 Critical care
3.2 Acute care in-patient
3.3 Extended care and skilled nursing facilities
3.4 Rehabilitation facility
3.5 Home care
3.6 Pulmonary rehabilitation program
3.7 Outpatient, ambulatory care
3.8 Pulmonary diagnostic laboratory
3.9 Sputum-induction room

DC 4.0 INDICATIONS:

4.1 The need to aid in the removal of retained secretions from central airways[3-6]—(the suggestion that FET at lower lung volumes may be effective in preferentially mobilizing secretions in peripheral airways while larger volumes facilitate movement in the central airways lacks validation).
4.2 The presence of atelectasis[3,7,8]
4.3 As prophylaxis against postoperative pulmonary complications[7]
4.4 As a routine part of bronchial hygiene in patients with cystic fibrosis,[2,4,6,9] bronchiectasis, chronic bronchitis,[3,10,11] necrotizing pulmonary infection, or spinal cord injury[12]
4.5 As an integral part of other bronchial hygiene therapies such as postural drainage therapy (PDT),[2,13] positive expiratory pressure therapy (PEP), and incentive spirometry (IS)
4.6 To obtain sputum specimens for diagnostic analysis

DC 5.0 CONTRAINDICATIONS:

Directed cough is rarely contraindicated. The contraindications listed must be weighed against potential benefit in deciding to eliminate cough from the care of the patient. Listed contraindications are relative.

5.1 Inability to control possible transmission of infection from patients suspected or known to have pathogens transmittable by droplet nuclei (eg, *M tuberculosis*)
5.2 Presence of an elevated intracranial pressure or known intracranial aneurysm
5.3 Presence of reduced coronary artery perfusion, such as in acute myocardial infarction[14]
5.4 Acute unstable head, neck, or spine injury

Manually assisted directed cough with pressure to the epigastrium may be contraindicated in presence of

5.5 increased potential for regurgitation/aspiration (eg, unconscious patient with unprotected airway)
5.6 acute abdominal pathology, abdominal aortic aneurysm, hiatal hernia, or pregnancy
5.7 a bleeding diathesis
5.8 untreated pneumothorax

Manually assisted directed cough with pressure to the thoracic cage may be contraindicated in presence of

5.9 osteoporosis, flail chest

DC 6.0 HAZARDS/COMPLICATIONS:

6.1 Reduced coronary artery perfusion[14]
6.2 Reduce cerebral perfusion leading to syncope or alterations in consciousness, such as, light-headedness or confusion,[15] vertebral artery dissection
6.3 Incontinence
6.4 Fatigue
6.5 Headache[15]
6.6 Paresthesia or numbness[15]
6.7 Bronchospasm[11]
6.8 Muscular damage or discomfort
6.9 Spontaneous pneumothorax, pneumomediastinum, subcutaneous emphysema
6.10 Cough paroxysms
6.11 Chest pain
6.12 Rib or costochondral junction fracture
6.13 Incisional pain, evisceration
6.14 Anorexia, vomiting, and retching
6.15 Visual disturbances including retinal hemorrhage[15]
6.16 Central line displacement[16]
6.17 Gastroesophageal reflux[17]

DC 7.0 LIMITATION OF METHOD:

7.1 Directed cough is of limited value in the obtunded, paralyzed, or uncooperative patient
7.2 The following clinical entities may compromise the effectiveness of a directed cough maneuver:
- **7.2.1** the presence of severe obstructive airways disease;
- **7.2.2** severe restrictive disease;
- **7.2.3** pain exacerbated by coughing (eg, incisional);
- **7.2.4** fear of pain;
- **7.2.5** neurologic, muscular, or skeletal abnormalities;[18,19]
- **7.2.6** systemic dehydration;
- **7.2.7** antitussives.

7.3 In patients with a bypassed upper airway or other condition that precludes the ability to effectively close the glottis, the effectiveness of the cough may be limited.
7.4 Thick, tenacious sputum may limit the effectiveness of these techniques and may require other supplemental strategies to optimize clearance of secretions.[3]

DC 8.0 ASSESSMENT OF NEED:

8.1 Spontaneous cough that fails to clear secretions from the airway
8.2 Ineffective spontaneous cough as judged by
- **8.2.1** clinical observation
- **8.2.2** evidence of atelectasis
- **8.2.3** results of pulmonary function testing

8.3 Postoperative upper abdominal or thoracic surgery patient[7]
8.4 Long-term care of patients with tendency to retain airway secretions

8.5 Presence of endotracheal or tracheostomy tube

DC 9.0 ASSESSMENT OF OUTCOME:

9.1 The presence of sputum specimen following a cough[4]
9.2 Clinical observation of improvement
9.3 Patient's subjective response to therapy
9.4 Stabilization of pulmonary hygiene in patients with chronic pulmonary disease and a history of secretion retention

DC 10.0 RESOURCES:

10.1 Equipment:
10.1.1 Material (such as tissues or gauze pads to cover mouth during cough and for disposal of expectorated secretions
10.1.2 Support devices for the patient with incisional pain or chest-wall pain during cough (eg, folded blanket, pillow, palmed hands,[20]or other devices)
10.1.3 Container for collecting expectorated sputum
10.1.4 Gloves, goggles, gown, and mask[21,22] (Mask should be designed to prevent transmission of droplet nuclei, eg, a disposable personal respirator[21])
10.1.5 Teaching materials and models

10.2 Personnel:
10.2.1 Level I personnel may be providers of service after Level II personnel have performed initial assessment of the patient and the first episode of training has been completed. (It is recommended that Level II personnel be credentialed, eg, CRTT, RRT, PT, or RN.) **Level I** personnel must demonstrate
10.2.1.1 proper use and limitation of equipment;
10.2.2.2 ability to assess patient's condition and response to therapy;
10.2.1.3 understanding of the procedures, indications, contraindications, and hazards of cough;
10.2.1.4 understanding of and compliance with Universal Precautions and infection control standards;[20,21]
10.2.1.5 Ability to instruct patient and family or caregiver in techniques and goals of therapy.
10.2.2 The **Level II** provider initially assesses the patient, determines the need for specific coughing techniques, and evaluates response to and effectiveness of cough. Level II personnel have all the skills of Level I providers plus
10.2.2.1 knowledge and understanding of patient's disease, goals, and limitation of cough;
10.2.2.2 ability to assess patient cough technique and perform initial instruction and treatment;
10.2.2.3 ability to make recommendations for changes in the patient's care, as indicated;
10.2.2.4 ability to teach the patient to respond appropriately in the home environment to changes in sputum quantity and quality.
10.2.3 The **subject** (patient) performing cough and FET without supervision should possess knowledge and skills related to
10.2.3.1 proper technique of related procedures;
10.2.3.2 possible hazards and complications;
10.2.3.3 technique modification in response to outcomes of therapy;
10.2.3.4 assessment and documentation of outcomes of cough with regard to sputum quantity, color, and other relevant characteristics;
10.2.3.5 appropriate response (according to established home prescription) to changes in sputum production.

DC 11.0 MONITORING:

Items from the following list should be chosen as appropriate for monitoring a patient's response to cough technique.

11.1 Patient response: pain, discomfort, dyspnea

11.2 Sputum expectorated following cough to note color consistency, odor, volume of sputum produced
11.3 Breath sounds
11.4 Presence of any adverse neurologic signs or symptoms following cough[15]
11.5 Presence of any cardiac dysrhythmias or alterations in hemodynamics following coughing
11.6 Measures of pulmonary mechanics, when indicated, may include vital capacity, peak inspiratory pressure, peak expiratory pressure, peak expiratory flow, and airway resistance

DC 12.0 FREQUENCY:

12.1 Cough procedures should be performed as frequently as needed. No data exist to support a specific frequency.
12.2 Cough should be performed as prophylactic measure by postoperative patients (recommended frequency—every 2-4 hours while awake).
12.3 FET as a therapeutic alternative for postural drainage therapy encompasses multiple maneuvers performed 3 to 4 times daily.
12.4 Cough procedures should be performed in conjunction with other forms of therapy to mobilize and remove secretions, during and at the conclusion of the therapy.

DC 13.0 INFECTION CONTROL ISSUES:

13.1 Cough is a source of droplet nuclei (aerosols) that can remain suspended in the air for hours and are associated with transmission of tuberculosis and other airborne pathogens. Care must be taken to minimize exposure of the health care worker and others.[22]

13.1.1 The single most effective step to reduce transmission of the droplet nuclei is to have the patient cover his or her mouth when coughing.

13.1.2 Local exhaust ventilation and personal protective equipment should be used to minimize exposure to airborne pathogens.[22]

13.1.3 Universal Precautions should be followed.[21]

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AARC Clinical Practice Guideline

Endotracheal Suctioning of Mechanically Ventilated Adults and Children with Artificial Airways

ETS 1.0 PROCEDURE:

Endotracheal suctioning of mechanically ventilated adults and children with artificial airways

ETS 2.0 DESCRIPTION:

Endotracheal suctioning is a component of bronchial hygiene therapy and mechanical ventilation and involves the mechanical aspiration of pulmonary secretions from a patient with an artificial airway in place. The procedure includes patient preparation, the suctioning event(s), and follow-up care.

2.1 Patient Preparation:

2.1.1 In preparation for the suctioning event, the patient ***should*** receive hyperoxygenation by the delivery of 100% oxygen for > 30 seconds prior to the suctioning event.[1,2] This may be accomplished by any of the following:

2.1.1.1 by adjusting the F_{IO_2} setting on the mechanical ventilator.

2.1.1.2 by use of a temporary oxygen-enrichment program available on many microprocessor ventilators.[3]

2.1.1.3 by manually ventilating the patient with an increased F_{IO_2}. (At least one study suggests that suctioning methods that use the ventilator for oxygenation and hyperinflation may be superior to use of the manual resuscitator.[4])

2.1.1.3.1 This technique has been shown to be ineffective for providing F_{DO_2} of 1.0.[5]

2.1.1.3.2 Practitioners should ensure that adequate PEEP levels are maintained while using this technique for patients requiring > 5 cm H_2O.

2.1.1.4 by the continuous or intermittent insufflation of oxygen through the suction catheter during the suction event[6-8]—Devices designed for this purpose may be difficult to use and are expensive.

2.1.1.5 It has been shown that delivery of increased tidal volumes is difficult while using this technique.[9]

2.1.2 In preparation for the suctioning event, the patient ***may be*** hyperventilated by delivery of an increased rate[1] and/or tidal volume. Hyperventilation may be accomplished by temporarily increasing the mandatory rate on the mechanical ventilator prior to the suctioning event or by increasing the rate of ventilation with a manual resuscitator. (Note: Care should be taken to maintain an adequate expiratory time to allow for complete exhalation of the delivered tidal volume.)[1]

2.1.3 In preparation for the suctioning event, the patient **may *be*** hyperinflated by the delivery of 'sigh' breaths prior to the suctioning event:

2.1.3.1 by increasing the ventilator-set tidal volume to exceed the set tidal volume,

2.1.3.2 by manually triggering preset sigh breaths on mechanical ventilators that are equipped with a sigh feature,[10] or

2.1.3.3 by manual ventilation with a resuscitation bag.

2.1.4 A closed-suction system may be used to facilitate continuous mechanical

ventilation and oxygenation during the suctioning event.[1]

2.1.5 A patient may be placed on a pulse oximeter to assess oxygenation during and following the procedure.

2.1.6 A patient may receive normal saline by instillation through the artificial airway to dilute and mobilize pulmonary secretions.[11,12] The value of instilling other solutions appears to be based on anecdotal reports.

2.2 The Suctioning Event: The placement of a suction catheter through the artificial airway into the trachea and the application of negative pressure as the catheter is being withdrawn. Sterile technique should be employed. Each pass of the suction catheter into the artificial airway is considered a suctioning event. The duration of each suctioning event should be approximately 10-15 seconds.[13] Suction pressure should be set as low as possible and yet effectively clear secretions. (Experimental data to support an appropriate maximum suction level are lacking. Some textbooks cite maximum safe limits of 100-150 torr but do not reference their recommendations.[14,15])

2.3 Follow-Up Care: Following the suctioning event,

2.3.1 the patient ***should be*** hyperoxygenated by delivery of 100% oxygen for ≥ 1 minute by the same technique(s) used to preoxygenate the patient.

2.3.2 the patient ***may be*** hyperventilated by increasing the respiratory rate and/or tidal volume by the same technique(s) used prior to suctioning.

2.3.3 the patient ***should be*** monitored for adverse reactions.

ETS 3.0 SETTING:

Endotracheal suctioning may be performed by properly trained persons in a wide variety of settings that include (but are not limited to):

3.1 Hospital
3.2 Extended care facility
3.3 Home
3.4 Outpatient clinic
3.5 Physician's office
3.6 Transport vehicle

ETS 4.0 INDICATIONS:

4.1 The need to remove accumulated pulmonary secretions as evidenced by one of the following:

4.1.1 Coarse breath sounds by auscultation or 'noisy' breathing

4.1.2 Increased peak inspiratory pressures during volume-controlled mechanical ventilation or decreased tidal volume during pressure-controlled ventilation.

4.1.3 Patient's inability to generate an effective spontaneous cough.

4.1.4 Visible secretions in the airway

4.1.5 Changes in monitored flow and pressure graphics

4.1.6 Suspected aspiration of gastric or upper airway secretions

4.1.7 Clinically apparent increased work of breathing

4.1.8 Deterioration of arterial blood gas values

4.1.9 Radiologic changes consistent with retention of pulmonary secretions

4.2 The need to obtain a sputum specimen to rule out or identify pneumonia or other pulmonary infection or for sputum cytology

4.3 The need to maintain the patency and integrity of the artificial airway

4.4 The need to stimulate a patient cough in patients unable to cough effectively secondary to changes in mental status or the influence of medication

4.5 Presence of pulmonary atelectasis or consolidation, presumed to be associated with secretion retention

ETS 5.0 CONTRAINDICATIONS:

Endotracheal suctioning is a necessary procedure for patients with artificial airways. Most contraindications are relative to the patient's risk of developing adverse reactions or worsening clinical condition as result of the procedure. When indicated, there is no absolute contraindication to endotracheal suctioning because the decision to ab-

stain from suctioning in order to avoid a possible adverse reaction may, in fact, be lethal.

ETS 6.0 HAZARDS/COMPLICATIONS:[1,2]

6.1 Hypoxia/hypoxemia[16-23]
6.2 Tissue trauma to the tracheal and/or bronchial mucosa[19,24]
6.3 Cardiac arrest[22,25]
6.4 Respiratory arrest[21]
6.5 Cardiac dysrhythmias[2,19,24]
6.6 Pulmonary atelectasis[17,19,21]
6.7 Bronchoconstriction/bronchospasm[19]
6.8 Infection (patient and/or caregiver)[19,26,27]
6.9 Pulmonary hemorrhage/bleeding[19,24]
6.10 Elevated intracranial pressure[28-30]
6.11 Interruption of mechanical ventilation[18]
6.12 Hypertension[31]
6.13 Hypotension[31]

ETS 7.0 LIMITATIONS OF METHOD:

Endotracheal suctioning is not a benign procedure, and operators should remain sensitive to possible hazards and complications and take all necessary precautions to ensure patient safety. Secretions in peripheral airways are not directly removed by endotracheal suctioning.

ETS 8.0 ASSESSMENT OF NEED:

Qualified personnel should assess the need for endotracheal suctioning as a routine part of a patient/ventilator system check.

ETS 9.0 ASSESSMENT OF OUTCOME:

9.1 Improvement in breath sounds
9.2 Decreased peak inspiratory pressure (PIP) with narrowing of PIP – $P_{plateau}$; decreased airway resistance or increased dynamic compliance; increased tidal volume delivery during pressure-limited ventilation
9.3 Improvement in arterial blood gas values (ABGs) or saturation as reflected by pulse oximetry (S_{pO_2})
9.4 Removal of pulmonary secretions

ETS 10.0 RESOURCES:

10.1 Necessary Equipment:
10.1.1 Vacuum source
10.1.2 Calibrated, adjustable regulator
10.1.3 Collection bottle and connecting tubing
10.1.4 Sterile disposable gloves
10.1.5 Sterile suction catheter of appropriate caliber. Diameter of the suction catheter should not exceed more than one half of the internal diameter of the artificial airway.[2,21,32] For selective main-stem suctioning, a curved-tip catheter may be helpful. The information related to the effectiveness of head turning for selective suctioning is inconclusive.
10.1.6 Sterile water and cup
10.1.7 Sterile normal saline, if instillation is desirable
10.1.8 Goggles, mask, and other appropriate equipment for Universal Precautions[33]
10.1.9 Oxygen source with a calibrated metering device
10.1.10 Manual resuscitation bag equipped with an oxygen enrichment device
10.1.11 Stethoscope

10.2 Optional Equipment:
10.2.1 EKG monitor
10.2.2 Pulse oximeter
10.2.3 Sterile sputum trap for culture specimen
10.2.4 Closed suction system
10.2.5 Oxygen insufflation device
10.2.5.1 Double-lumen suction catheter for continuous oxygen insufflation[4,7,16]
10.2.5.2 Control valve device to alternate suction and oxygen insufflation to the suction catheter[7,32]

10.3 Personnel: Personnel responsible for performing endotracheal suctioning should demonstrate the following—
10.3.1 Knowledge of proper use and assembly of all equipment used
10.3.2 Ability to recognize abnormal breath sounds by auscultation
10.3.3 Knowledge and understanding of the patient's history, disease process, and goals of treatment

10.3.4 Knowledge and understanding of basic physiology and pathophysiology
10.3.5 Knowledge and understanding of ventilation, mechanical ventilators, and their alarm systems
10.3.6 Knowledge and understanding of all artificial airways and adjuncts used
10.3.7 Ability to monitor vital signs, assess the patient's condition, and appropriately respond to complications or adverse reactions to the procedure
10.3.8 Ability to modify techniques and equipment in response to complications or adverse reactions
10.3.9 Knowledge of basic EKG interpretation
10.3.10 Ability to assess the need for and provide cardiopulmonary resuscitation
10.3.11 Ability to evaluate and document the effectiveness and patient response to the procedure
10.3.12 Knowledge and understanding of the CDC guidelines for Universal Precautions
10.3.13 Knowledge of signs and symptoms of decreased cardiac output, oxygenation, and perfusion
10.3.14 Ability to teach lay-persons the procedure for home and extended care
10.3.15 Lay-persons responsible for endotracheal suctioning outside the hospital should demonstrate:
- **10.3.15.1** Knowledge, skill, and understanding of the assembly, use, maintenance, and cleaning of all equipment used
- **10.3.15.2** Ability to assess the need for and patient response to the procedure
- **10.3.15.3** Ability to perform the proper suctioning technique
- **10.3.15.4** Ability to assess the need for and provide cardiopulmonary resuscitation

ETS 11.0 MONITORING:

The following should be monitored prior to, during, and after the procedure:

- **11.1** Breath sounds
- **11.2** Oxygen saturation
  - **11.2.1** Skin color
  - **11.2.2** Pulse oximeter, if available
- **11.3** Respiratory rate and pattern
- **11.4** Hemodynamic parameters
  - **11.4.1** Pulse rate
  - **11.4.2** Blood pressure, if indicated and available
  - **11.4.3** EKG, if indicated and available
- **11.5** Sputum characteristics
  - **11.5.1** Color
  - **11.5.2** Volume
  - **11.5.3** Consistency
  - **11.5.4** Odor
- **11.6** Cough effort
- **11.7** Intracranial pressure, if indicated and available
- **11.8** Ventilator parameters
  - **11.8.1** Peak inspiratory pressure and plateau pressure
  - **11.8.2** Tidal volume
  - **11.8.3** Pressure, flow, and volume graphics, if available
  - **11.8.4** F_{IO_2}
- **11.9** Arterial blood gases, if indicated and available

ETS 12.0 FREQUENCY:

Endotracheal suctioning should be performed whenever clinically indicated, with special consideration for the potential complications associated with the procedure. Endotracheal suctioning may be required at some minimum frequency in order to maintain the patency of the artificial airway used.

ETS 13.0 INFECTION CONTROL:

- **13.1** CDC Guidelines for Universal Precautions should be adhered to.[33]
- **13.2** All equipment and supplies should be appropriately disposed of or disinfected.

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AARC Clinical Practice Guideline

In-Vitro pH and Blood Gas Analysis and Hemoximetry

BGA 1.0 PROCEDURE:

In-vitro blood gas and pH analysis and hemoximetry (ie, CO-oximetry)

BGA 2.0 DESCRIPTION:

Analysis of arterial and/or mixed venous blood provides information concerning the oxygenation, ventilatory, and acid-base status of the subject from whom the specimen was obtained. (Capillary, peripheral venous, and umbilical venous samples may provide limited information.) Those variables that are generally measured are the partial pressures for carbon dioxide and oxygen (P_{CO_2} and P_{O_2}), hydrogen ion concentration (pH), and, if available, the concentration of total hemoglobin (tHb), oxyhemoglobin saturation (O_2Hb), and saturations of the dyshemoglobins (carboxyhemoglobin, or COHb, and methemoglobin, or metHb),[1-6] and other calculated or derived values such as plasma bicarbonate and base excess/deficit.

BGA 3.0 SETTING:

Analysis may be performed by trained individuals[4,5,7] in a variety of settings including, but not limited to,

3.1 Hospital laboratories
3.2 Hospital emergency and patient-care areas
3.3 Clinic laboratories
3.4 Laboratories in physicians' offices[8]

BGA 4.0 INDICATIONS:

Indications for blood gas and pH analysis and hemoximetry include

4.1 The need to evaluate the adequacy of a patient's ventilatory (P_{aCO_2}), acid-base (pH and P_{aCO_2}), and/or oxygenation (P_{aO_2} and O_2Hb) status, and the oxygen-carrying capacity (P_{aO_2}, O_2Hb, tHb, and dyshemoglobin saturations)[1,2,5,6] and intrapulmonary shunt ($\dot{Q}_{sp}/\dot{Q}_t$).
4.2 The need to quantitate the response to therapeutic intervention (eg, supplemental oxygen administration, mechanical ventilation) and/or diagnostic evaluation (eg, exercise desaturation).[1-3]
4.3 The need to monitor severity and progression of documented disease processes.[1,2,6]

BGA 5.0 CONTRAINDICATIONS:

Contraindications to performing pH-blood gas analysis and hemoximetry include:

5.1 An improperly functioning analyzer[4]
5.2 An analyzer that has not had functional status validated by analysis of commercially prepared quality control products or tonometered whole blood[4,9-12] or has not been validated through participation in a proficiency testing program(s)[7,9,11-15]
5.3 A specimen that has not been properly anticoagulated[4,5,12,16]
5.4 A specimen containing visible air bubbles[1,4,5]
5.5 A specimen that has been kept at room temperature > 5 min before analysis, or has not been properly stored on ice-water slush for analysis in ≤ 2 hours, or has been stored on ice-water slush for > 2 hours.[1,4-6]
5.6 An incomplete requisition that precludes adequate interpretation and documentation of results—requisitions should contain

5.6.1 Patient's name or other unique identifier such as medical record number; date and time of sampling
5.6.2 Name of requesting physician or authorized individual
5.6.3 Tests to be performed
5.6.4 Sample source (arterial line, central venous catheter, peripheral artery)
5.6.5 F_{IO_2} or oxygen flowrate for patient on supplemental oxygen
5.6.6 Ventilator settings for mechanically ventilated patients (tidal volume, respiratory rate, F_{IO_2}, mode)
5.6.7 Signature of person who obtained sample[4,5,7,12]

It may also be useful to note body temperature, activity level, and working diagnosis. Test requisition should be electronically generated or written and must be signed by the person ordering the test. Oral requests must be supported by written authorization within 30 days.[7]

5.7 An inadequately labeled specimen lacking the patient's full name or other unique identifier (eg, medical record number), date, and time of sampling[5,12]

BGA 6.0 HAZARDS/COMPLICATIONS:

6.1 Infection of specimen handler from blood (HIV, hepatitis B, other blood-borne pathogens)[8,10,12,17]
6.2 Inappropriate patient medical treatment based on improperly analyzed blood specimen,[18] or from analysis of an unacceptable specimen, or from incorrect reporting of results.

BGA 7.0 LIMITATIONS OF PROCEDURE/ VALIDATION OF RESULTS:

7.1 Limitations of technique or methodology that limit value of the procedure include
7.1.1 Sample clotting due to improper anticoagulation or improper mixing[1,12,19]
7.1.2 Sample contamination by:
7.1.2.1 air;
7.1.2.2 improper anticoagulant and/or anticoagulant concentration;
7.1.2.3 saline/fluids (specimen obtained via an indwelling catheter);
7.1.2.4 inadvertent sampling of systemic venous blood;
7.1.2.5 a time delay in sample analysis (ie, sample allowed to remain at room-temperature longer than 5 minutes or at temperature ≤ 5 °C for > 2 hours, or if P_{aO_2} is expected to be ≥ 100 torr and specimen is not analyzed within 5 minutes even if maintained at a temperature ≤ 5 °C[1,4,5,15,18]—P_{aO_2} in samples drawn from subjects with very high leukocyte counts can decrease very rapidly. Immediate chilling and analysis is necessary.[20,21]
7.1.2.6 insufficient clearance of analyzer calibration gases and previous waste or flushing solution(s).[12]
7.1.2.7 Hyperlipidemia causes problems with analyzer membranes.[12]
7.1.3 Appropriate sample size is determined by the type of anticoagulant[1,2,4,12] and the sample requirements of the analyzer(s).[10]
7.1.4 Total hemoglobin concentration measurement is dependent upon homogeneous mixture of specimen, anticoagulant concentration-to-specimen-size ratio, and contamination of specimen by analyzer solutions or calibration gases.
7.1.5 Some calculated values may be in error (eg, calculated S_{aO_2} may not reflect O_2Hb in the presence of COHb, and/or metHb and changes in 2,3 DPG concentration).
7.1.6 Arterialized capillary samples may be adequate to assess acid-base disorders but may not adequately reflect patient oxygenation.
7.2 Results of analysis can be considered valid if
7.2.1 analytic procedure conforms to recommended, established guidelines[10,12] and follows manufacturer's recommendations;
7.2.2 results of pH-blood gas analysis fall within the calibration range of the analyzer(s) and quality control product ranges;[7] If a result outside of the usual calibration

range is obtained (eg, P_{aO_2} measured as 250 torr, but analyzer calibrated to 140 torr), the analyzer should be recalibrated to accommodate this unusual value (using "calibration override" function and high- or 100%-oxygen standard gas).

7.2.3 laboratory and personnel are in compliance with quality control and recognized proficiency testing programs[7,9,10,12]

7.3 If questionable results are obtained and are consistent with specimen contamination:

7.3.1 The labeling of the blood sample container should be rechecked for patient's full name, medical record number (patient I.D.), date and time of acquisition and measured F_{IO_2} (or supplemental oxygen liter flow);[4,5,12]

7.3.2 The residual specimen should be reanalyzed (preferably on a separate analyzer);

7.3.3 If discrepancy cannot be resolved, an additional sample should be obtained;

7.3.4 Discarded samples should be logged with reason for discarding.[7]

BGA 8.0 ASSESSMENT OF NEED:

The presence of indications (BGA 4.0) in the subject to be tested supports the need for sampling and analysis.

BGA 9.0 ASSESSMENT OF TEST QUALITY:

9.1 Blood gas-pH analysis and hemoximetry are beneficial only if no preanalytical error has occurred (CPG: Sampling for Arterial Blood Gas Analysis, Respir Care 1992;37:913-917), and the analysis was

9.1.1 performed with a properly functioning analyzer (validated by quality control data);[7,9,12]

9.1.2 carried out according to an established proven protocol, conforming to manufacturer recommendations;[10,12]

9.2 The results should validate or contradict the patient's clinical condition (ie, the basis for ordering the test).[22]

9.3 Documentation of results, therapeutic intervention (or lack of), and/or clinical decisions based upon the pH-blood gas measurements should be available in the patient's medical record and/or be otherwise readily accessible (eg, at the testing area) for at least 2 years.[7]

9.4 Results should be interpreted by a physician in the light of the clinical question to be answered (eg, exercise desaturation).

BGA 10.0 RESOURCES:

Federal Regulations,[7] stipulate requirements relative to personnel (levels of education and training), documentation procedures and equipment. Blood gas instrumentation is classified as being either moderately or highly complex. Persons performing blood gas analysis should be conversant with the Federal regulations (so-called CLIA'88) and appropriately qualified.[7]

10.1 Recommended Equipment:

10.1.1 Automated or semiautomated pH-blood gas analyzer with related calibration gases, electrodes, membranes, electrolytes, and accessories[7,10,12]

10.1.2 Fixed, multiple wavelength spectrophotometer (hemoximeter, or CO-oximeter)[8] or other device for determining total hemoglobin and its components

10.1.3 Protective eye wear as necessary and outer wear, protective gloves, impenetrable needle container, face mask and/or face-shield.[23]

10.2 Personnel:

The following recommendations are for tests of moderate complexity, as designated by CLIA '88.[7] Persons at either of the levels described should perform blood gas-pH analysis under the direction and responsibility of a laboratory director and technical consultant (may be the same individual) who possesses a MD, PhD, master's or baccalaureate degree and who has specific training in blood gas analysis and interpretation.[7]

10.2.1 Level I: Personnel should be specifically trained in pH-blood gas analysis, oxygen delivery devices, and

related equipment, record keeping, and hazards and sources of specimen and handler contamination(s) associated with sampling and analysis. Such persons should be, at minimum, high school graduates (or equivalent) with strong backgrounds in mathematics, and preferably with one or more years of college courses in the physical and biological sciences. Such persons must have *documented* training and *demonstrated* proficiency in pH-blood gas analysis, preventive maintenance, troubleshooting, instrument calibration, and awareness of the factors that influence test results, and the skills required to verify the validity of test results through the evaluation of quality-control sample values, prior to analyzing patient specimens and reporting results.[7,24] Performance of pH-blood gas analysis must be supervised by a Level II individual.

10.2.2 Level II: Level II personnel supervise Level I personnel and are health care professionals specifically trained (with proven, documented proficiency) in all aspects of blood gas analysis and hemoximetry:

10.2.2.1 quality control, quality assurance, and proficiency testing;

10.2.2.2 operation and limitations, including instrument trouble-shooting and appropriate corrective measures.

10.2.2.3 Level II personnel should be cognizant of various means for specimen collection and the causes and impact of preanalytical and instrument error(s).

10.2.2.4 Level II personnel should be trained in patient assessment, acid-base and oxygenation disorders, diagnostic and therapeutic alternatives. A baccalaureate, or higher, degree in the sciences or substantial experience in pulmonary function technology is preferred. Although, 2 years of college in biological sciences and mathematics, plus 2 years of training and experience, or equivalent may be substituted for personnel supervising arterial pH-blood gas analysis. A credential (MT, MLT, CRTT, RRT, CPFT, RPFT) is strongly recommended.[7,24]

BGA 11.0 MONITORING:

The following aspects of analysis should be monitored and corrective action taken as indicated:

11.1 presence of air bubbles or clots in specimen, with evacuation prior to mixing and sealing syringe;[1,4,5,12]

11.2 assurance that a solid/continuous sample is aspirated (or injected) into analyzer and that all of the electrodes are covered by the sample (confirmed by direct viewing of sample chamber if possible[10]);

11.3 assurance that 8-hour quality control and calibration procedures have been completed and that instrumentation is functioning properly prior to patient sample analysis;[4,7,9,12]

11.4 assurance that specimen was properly labeled, iced, and analyzed within an acceptable period of time;[4,5,12]

11.5 participation in an accredited (recognized) proficiency testing program (pH, P_{CO_2}, P_{O_2}, and tHb proficiency testing is mandatory for all laboratories).[7] Other reported hemoximeter variables (particularly O_2Hb, COHb, MetHb) need to have accuracy and reliability evaluated at least twice each year.

11.6 As part of any quality assurance program, indicators must be developed to monitor those areas addressed in Sections 5.0, 7.0, 10.0, 12.0, and 13.0.

11.6.1 There must be evidence of active review of quality control, proficiency testing, and physician alert, or 'panic values,'on a level commensurate with the number of tests performed.

11.6.2 Personnel who do not meet acceptable performance thresholds should not be allowed to independently participate further, until they have received remedial instruction and have been re-evaluated.

BGA 12.0 FREQUENCY:

12.1 The frequency with which pH-blood gas analysis is repeated (on different samples from the same patient) should depend on the clinical

status of the patient and not on an arbitrarily designated time or frequency.

12.2 The frequency of reanalysis of single specimens (eg, verification of results) depends on

12.2.1 laboratory protocol

12.2.2 technician/technologist suspicion that preliminary results may not reflect the clinical status of patient (ie, preanalytical or instrument error may be present)[10]

BGA 13.0 INFECTION CONTROL:

13.1 Universal Precautions must be applied to all handling of blood and/or contaminated instruments, and collection devices.[10,12,25]

13.2 Disposal of blood samples and collection devices must conform to institution policy, recommendations, or guidelines[10,23]

13.3 Cleaning and maintenance procedures must adhere to CDC guidelines & manufacturers' recommendations.

13.4 Blood spills should be handled according to procedures developed in accordance with CDC and OSHA directives.[21,25]

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AARC Clinical Practice Guideline

Single-Breath Carbon Monoxide Diffusing Capacity

DCO 1.0 PROCEDURE:

Single-breath diffusing capacity for carbon monoxide ($D_{LCO\text{-}sb}$). This test is also sometimes referred to as the transfer factor for carbon monoxide.

DCO 2.0 DESCRIPTION/DEFINITION:

Diffusing capacity is a test in which the subject inspires a gas containing carbon monoxide and one or more tracer gases in order to determine the gas exchange capacity of the lungs. Although several different methods of measuring D_{LCO} have been described, the most commonly used technique is the single-breath maneuver or $D_{LCO\text{-}sb}$. For purposes of this guideline, recommendations associated with the $D_{LCO\text{-}sb}$ are referenced.[1] Many of these standards apply indirectly to other methods of measuring diffusing capacity.

2.1 D_{LCO} is usually expressed in mL CO · min^{-1} · $torr^{-1}$ (STPD).
2.2 The alveolar volume (V_A) at which the $D_{LCO\text{-}sb}$ is measured is also commonly reported; the units for the V_A are liters at body temperature and pressure, saturated with water vapor (BTPS).
2.3 The ratio of D_{LCO} to V_A is also commonly reported as the D_L/V_A or simply D/V_A.

DCO 3.0 SETTINGS:

3.1 Pulmonary function laboratories
3.2 Cardiopulmonary laboratories
3.3 Clinics
3.4 Physicians' offices

DCO 4.0 INDICATIONS:

Tests of diffusing capacity may be indicated in

4.1 Evaluation and follow-up of parenchymal lung diseases associated with dusts (eg, asbestos) or drug reactions (eg, amiodarone) or related to sarcoidosis;[1,2]
4.2 Evaluation and follow-up of emphysema and cystic fibrosis;[3,4]
4.3 Differentiating among chronic bronchitis, emphysema, and asthma in patients with obstructive patterns;
4.4 Evaluation of pulmonary involvement in systemic diseases (eg, rheumatoid arthritis, systemic lupus erythematosus);
4.5 Evaluation of cardiovascular diseases (eg, primary pulmonary hypertension, pulmonary edema, acute or recurrent thromboembolism);[5,6]
4.6 Prediction of arterial desaturation during exercise in chronic obstructive pulmonary disease;[7,8]
4.7 Evaluation and quantification of disability associated with interstitial lung disease;[9]
4.8 Evaluation of the effects of chemotherapy agents, or other drugs known to induce pulmonary dysfunction;
4.9 Evaluation of hemorrhagic disorders.[10]

DCO 5.0 CONTRAINDICATIONS:

Relative contraindications to performing a diffusing capacity test are

5.1 Mental confusion or muscular incoordination preventing the subject from adequately performing the maneuver;
5.2 A large meal or vigorous exercise immediately before the test;[1]

5.3 Smoking within 24 hours of test administration—smoking may have a direct effect on D_{LCO} independent of the effect COHb.[11]

DCO 6.0 HAZARDS/COMPLICATIONS:

6.1 Single breath D_{LCO} requires breath holding at TLC; some patients may perform either a Valsalva (high intrathoracic pressure) or Müller (low intrathoracic pressure) maneuver. Either of these can result in alteration of venous return to the heart.

6.2 Transmission of infection is possible via improperly cleaned mouthpieces or as a consequence of the inadvertent spread of droplet nuclei or body fluids (patient to patient or patient to technologist).

DCO 7.0 LIMITATIONS OF METHODOLOGY/VALIDATION OF RESULTS:

7.1 Limitations of the typical methods used for D_{LCO} include

7.1.1 D_{LCO} should be corrected for hemoglobin level according to the recommendations of the American Thoracic Society (ATS) using the Cotes method.[12]

7.1.2 D_{LCO} should be corrected for the effects of COHb present in the subject's blood for purpose of interpretation.[1]

7.1.3 D_{LCO} increases with increasing altitude and appropriate correction for the alveolar or inspired oxygen pressures are recommended.[1]

7.1.4 A 4-minute minimum interval should elapse between subsequent maneuvers to allow test gas to be eliminated from the lungs.[1]

7.1.5 D_{LCO} varies with body position; the upright seated position is recommended.

7.1.6 Abnormal breath holding maneuvers (Valsalva or Müller) alter D_{LCO}.[13]

7.1.7 Other factors that may alter measurement of D_{LCO} include recent alcohol consumption,[14] vigorous exercise, smoking, diurnal variation, and bronchodilators.[15]

7.1.8 Pregnancy (1st trimester only) is associated with an increase in D_{LCO}.[16] Menstruation may also influence D_{LCO}.[17]

7.1.9 Breath-hold time should be calculated using the methods of Jones-Meade or Ogilvie.[1] Other methods may produce significantly different results.

7.2 Large interlaboratory differences in measured D_{LCO} and in percent of predicted D_{LCO} have been observed[18,19] and are attributed to variations in testing techniques and computational algorithms and errors in gas analysis. The choice of equipment may also influence the measured D_{LCO}.[19,20]

7.3 Choice of reference equations may affect the final interpretation of measured D_{LCO} values.[21]

7.4 Validation of the testing technique/equipment may include but is not limited to

7.4.1 Volume accuracy of spirometer (should meet all ATS recommendations) and maintain accuracy with varying gas concentrations.

7.4.2 Gas analyzers should have a 2-point calibration before each test, and linearity should be formally checked at least every 6 months.[22]

7.4.3 Timing device should be checked quarterly.[22]

7.4.4 Chemical absorbers (for CO_2 and H_2O) should be replaced when exhausted.

7.4.5 Normal standard subjects may be used to establish intrasubject coefficient of variation and to serve as a quality control population.

DCO 8.0 ASSESSMENT OF NEED (see Section 4.0 Indications)

DCO 9.0 ASSESSMENT OF TEST QUALITY:

Individual test maneuvers and results, should be evaluated according to the ATS recommendations.[1] In particular,

9.1 The inspiratory volume should exceed 90% of the largest previously measured vital capacity (FVC or VC).

9.2 Breath-hold time should be between 9 and 11 seconds, with a rapid inspiration.

9.3 The washout volume (dead space) should be 0.75 to 1.00 L, or 0.50 L if the subject's VC is less than 2.0 L. If a washout volume other than 0.75-1.00 is used, it should be noted.
9.4 Two or more acceptable tests should be averaged; the maneuvers should be reproducible to within 10% or 3 mL $CO \cdot min^{-1} \cdot torr^{-1}$, whichever is greater.
9.5 The subject should have refrained from smoking for 24 hours prior to the test.
9.6 Corrections for Hb, COHb should be included as noted above (Sections 7.1.1 and 7.1.2); correction for tests performed at altitude is recommended. If Hb correction is made, both the corrected and uncorrected D_{LCO} values should be reported.

DCO 10.0 RESOURCES:

10.1 Equipment:

10.1.1 Volume measuring device must meet or exceed ATS recommendations.

10.1.2 Appropriate gas analyzers dependent on the methodology employed; certified calibration gases for use before each series of measurements.

10.1.3 CO-oximeter for analysis of total Hb and COHb is strongly recommended.

10.2 Personnel: Diffusing capacity tests should be performed under the direction of a physician trained in pulmonary function testing and may be performed by persons who meet criteria for either Level I or Level II. The value of D_{LCO} results can be compromised when the test is performed by inadequately trained personnel.

10.2.1 Level I: Personnel performing D_{LCO} should be a high school graduate or equivalent with a demonstrated ability to perform basic pulmonary function studies such as spirometry (and $D_{LCO\text{-}sb}$). Level I personnel should perform D_{LCO} tests *only* under the supervision of a Level II individual or a physician.

10.2.2 Level II: Personnel supervising D_{LCO} testing should have formal education and training (as a part of a program in respiratory therapy or pulmonary function technology or 2 years of college work in biological sciences and mathematics) and 2 or more years performing spirometry, lung volumes, and diffusing capacity tests.[23] One or more of the following credentials is recommended: CRTT, RRT, CPFT, RPFT.

DCO 11.0 MONITORING: (Also see Section 9.0 Assessment of Test Quality)

11.1 The final report should contain a statement about test quality.

11.2 The final report should contain the D_{LCO}, the corrected D_{LCO} (Hb, COHb, altitude), and the Hb value used for correction. The alveolar volume (V_A) and D_L/V_A (ie, the ratio of diffusing capacity to the lung volume at which the measurement was made) may be included in the report. These values are helpful for purposes of interpretation.

DCO 12.0 FREQUENCY:

How often D_{LCO} measurements should be repeated depends on the clinical question(s) to be answered.

DCO 13.0 INFECTION CONTROL:

Diffusing capacity tests are relatively safe procedures, but the possibility of cross-contamination exists, either from the patient-patient or patient-technologist interface.[24]

13.1 Universal Precautions[25,26] (as published by the Centers for Disease Control) should be applied in all instances in which there is evidence of contamination with blood (eg, mouthpieces). Although Universal Precautions do not apply to saliva or mucus unless it contains blood, it should be noted that other potentially hazardous organisms may be present in these fluids even in the absence of blood, and the appropriate use of barriers and hand washing are recommended.

13.2 Due to the nature of the D_{LCO} maneuvers and the likelihood of coughing when the test is performed by subjects with known or suspected active infection with *M tuberculosis* or other airborne organisms, the following precautions are recommended:

13.2.1 The room in which the D_{LCO} test is performed should meet or exceed the recommendations of U.S. Public Health Service[24] for air changes and ventilation. Ideally, an area in the testing department should be specially ventilated for isolation patients. If this is not possible, the patient should be returned to the isolation room as soon as possible.

13.2.2 Pulmonary function technologists performing procedures on patients with potentially infectious airborne diseases should wear a personal respirator that meets OSHA recommendations, especially if the testing itself induces cough.[27]

13.3 The mouthpiece, tubing, and any parts of the system that come into contact with the subject should be disposable or sterilized between patients. If sterilization is not feasible, then high-level disinfection should be performed. It is unnecessary to routinely clean the interior surface of the spirometer.

13.4 Bacteria filters may be used in circuits that allow rebreathing, although their efficacy is not well documented. However, such filters may impose added resistance during inspiration or expiration and affect the timing of the D_{LCO} maneuver. If a filter is used, the filter dead space volume should be considered in the calculation of D_{LCO} and V_A.

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AARC Clinical Practice Guideline

Use of Positive Airway Pressure Adjuncts to Bronchial Hygiene Therapy

PAP 1.0 PROCEDURE:

Positive airway pressure (PAP) adjuncts are used to mobilize secretions and treat atelectasis and include continuous positive airway pressure (CPAP), positive expiratory pressure (PEP), and expiratory positive airway pressure (EPAP).

Cough or other airway clearance techniques are essential components of PAP therapy when the therapy is intended to mobilize secretions.[1-3]

PAP 2.0 DEFINITION/DESCRIPTION:

During CPAP therapy, the patient breathes from a pressurized circuit against a threshold resistor (water-column, weighted, or spring loaded) that maintains consistent preset airway pressures from 5 to 20 cm H_2O during both inspiration and expiration.[4-13] (By strict definition, CPAP is any level of above-atmospheric pressure.) CPAP requires a gas flow to the airway during inspiration that is sufficient to maintain the desired positive airway pressure.

During PEP therapy, the patient exhales against a fixed-orifice resistor, generating pressures during expiration that usually range from 10 to 20 cm H_2O.[14-24] PEP does not require a pressurized external gas source.

During EPAP therapy the patient exhales against a threshold resistor, generating preset pressures of 10 to 20 cm H_2O.[25-27] EPAP does not require a pressurized external gas source.

EPAP utilizing threshold resistors does not produce the same mechanical or physiologic effects that PEP does when a fixed orifice resistor is used.[28] Further study is necessary to determine how these differences affect clinical outcome.

PAP 3.0 SETTINGS:

3.1 Critical care
3.2 Acute care inpatient
3.3 Extended-care and transitional-care facilities
3.4 Home care
3.5 Outpatient

PAP 4.0 INDICATIONS:

4.1 To reduce air trapping in asthma and COPD[16,29-31]
4.2 To aid in mobilization of retained secretions (in cystic fibrosis and chronic bronchitis)[14,15,17-24,32,33]
4.3 To prevent or reverse atelectasis[6-13,34-36]
4.4 To optimize delivery of bronchodilators in patients receiving bronchial hygiene therapy[37,38]

PAP 5.0 CONTRAINDICATIONS:

Although no absolute contraindications to the use of PEP, CPAP, or EPAP mask therapy have been reported,[4,39] the following should be carefully evaluated before a decision is made to initiate PAP mask therapy.

5.1 Patients unable to tolerate the increased work of breathing (acute asthma, COPD)
5.2 Intracranial pressure (ICP) > 20 mm Hg
5.3 Hemodynamic instability[4]
5.4 Recent facial, oral, or skull surgery or trauma[4]
5.5 Acute sinusitis[39]

5.6 Epistaxis
5.7 Esophageal surgery
5.8 Active hemoptysis[39]
5.9 Nausea
5.10 Known or suspected tympanic membrane rupture or other middle ear pathology
5.11 Untreated pneumothorax

PAP 6.0 HAZARDS/COMPLICATIONS:

6.1 Increased work of breathing[4] that may lead to hypoventilation and hypercarbia
6.2 Increased intracranial pressure
6.3 Cardiovascular compromise
  6.3.1 myocardial ischemia
  6.3.2 decreased venous return[4]
6.4 Air swallowing,[4] with increased likelihood of vomiting and aspiration
6.5 Claustrophobia[4]
6.6 Skin break down and discomfort from mask[4]
6.7 Pulmonary barotrauma[4]

PAP 7.0 LIMITATIONS OF METHOD:

7.1 PAP therapies for bronchial hygiene require spontaneously breathing patients.
7.2 CPAP is an equipment-intensive procedure requiring an external positive pressure gas source or compressor and considerable training of personnel for proper setup and maintenance. These factors make CPAP more expensive and less portable than other PAP alternatives.

PAP 8.0 ASSESSMENT OF NEED:

The following should be assessed together to establish a need for PAP therapy:

8.1 Sputum retention not responsive to spontaneous or directed coughing
8.2 History of pulmonary problems treated successfully with postural drainage therapy
8.3 Decreased breath sounds or adventitious sounds suggesting secretions in the airway
8.4 Change in vital signs—increase in breathing frequency, tachycardia
8.5 Abnormal chest radiograph consistent with atelectasis, mucus plugging, or infiltrates
8.6 Deterioration in arterial blood gas values or oxygen saturation

PAP 9.0 ASSESSMENT OF OUTCOME:

9.1 *Change in sputum production*—if PEP does not increase sputum production in a patient who produces > 30 mL/day of sputum without PEP, the continued use of PEP may not be indicated.
9.2 *Change in breath sounds*—with effective therapy, breath sounds may clear or the movement of secretions into the larger airways may cause an increase in adventitious breath sounds. The increase in adventitious breath sounds is often a marked improvement over no (or diminished) breath sounds. Note any effect that coughing may have had on the breath sounds.
9.3 *Patient subjective response to therapy*—the caregiver should ask the patient how he or she feels before, during, and after therapy. Feelings of pain, discomfort, shortness of breath, dizziness, and nausea should be considered in modifying and stopping therapy. Improved ease of clearing secretions and increased volume of secretions during and after treatments support continuation.
9.4 *Change in vital signs*—moderate changes in respiratory rate and/or pulse rate are expected. Bradycardia, tachycardia, increasingly irregular pulse, or a drop or dramatic increase in blood pressure are indications for stopping therapy.
9.5 *Change in chest radiograph*—resolution or improvement of atelectasis and localized infiltrates may be slow or dramatic.
9.6 *Change in arterial blood gas values or oxygen saturation*—normal oxygenation should return as atelectasis resolves.

PAP 10.0 RESOURCES:

10.1 Equipment:
  10.1.1 *PEP*—Fixed orifice resistor capable of developing 10 to 20 cm H_2O pressure during passive expiration, with one-way valves allowing unobstructed inspiration.[39,41,42]
  10.1.2 *CPAP*—Threshold resistor capable of developing 5 to 20 cm H_2O pressure, with a source of gas flow sufficient to

maintain the desired level of pressure during inspiration, at desired F_{IO_2} (requiring flowrater or blender, reservoir bag on inspiratory line, or an adjustable demand valve)

10.1.3 *EPAP*—Threshold resistor capable of developing pressures of 10 to 20 cm H_2O, with a one-way valve that allows gas at ambient pressure to enter airway on inspiration and directs exhaled gas through the threshold resistor[28]

10.1.4 Transparent mask or mouthpiece[12,39]

10.1.5 Manometer for initial adjustments of resistor size and/or gas flow[39]

10.1.6 Tissues and emesis basin or container for collecting or disposing of expectorated sputum

10.1.7 Gloves, goggles, gown, and mask

10.2 Personnel: A spectrum of education and skill levels is required for personnel who administer PEP, CPAP, or EPAP therapy. Different clinical situations warrant the degree of training necessary to provide optimal respiratory care:

10.2.1 Level-I personnel are responsible for ongoing assessment and care of unstable patients. Their demonstrated skills and knowledge should include

10.2.1.1 proper use and limitations of equipment;

10.2.1.2 ability to assess patient condition and response to therapy;

10.2.1.3 performance of physical examination (auscultation and vital signs);

10.2.1.4 understanding of effects of increased expiratory pressure on ventilation, perfusion, and sputum mobilization;

10.2.1.5 understanding of procedures, indications, and contraindications, and hazards for PEP, CPAP, and EPAP;

10.2.1.6 ability to demonstrate diaphragmatic breathing and relaxation, and to direct coughing;

10.2.1.7 ability to monitor effects of and subject response to changes in expiratory airway pressure;

10.2.1.8 understanding of and compliance with Universal Precautions and infection control standards related to cleaning equipment, maintaining equipment, and handling of secretions.

10.2.2 Level-II personnel should possess all Level-I skills and knowledge plus

10.2.2.1 ability to perform initial assessment of patient, initiate therapy and assess patient response and tailor therapy to patient needs.

10.2.2.2 ability to negotiate care plan and modifications with physician and health care team

10.2.2.3 ability to instruct patient, family, or caregiver in goals of therapy and

10.2.2.3.1 proper technique for administration,

10.2.2.3.2 proper use of equipment,

10.2.2.3.3 cleaning of equipment,

10.2.2.3.4 breathing patterns and cough techniques,

10.2.2.3.5 modification of technique in response to adverse reactions,

10.2.2.3.6 modification of duration or frequency in response to severity of symptoms.

10.2.3 Level III: Self-administration of PEP, EPAP, or CPAP—the patient who is to self-administer treatment should demonstrate

10.2.3.1 proper technique for administration,

10.2.3.2 proper use of equipment,

10.2.3.3 appropriate breathing patterns and cough techniques,

10.2.3.4 ability to modify technique in response to adverse reactions,

10.2.3.5 ability to modify duration or frequency in response to severity of symptoms.

PAP 11.0 MONITORING:

Items from the following list should be chosen as is appropriate for monitoring a specific patient's response to PAP.

11.1 Patient subjective response—pain, discomfort, dyspnea, response to therapy
11.2 Pulse rate and cardiac rhythm (if EKG is available)
11.3 Breathing pattern and rate, symmetrical lateral costal expansion, synchronous thoracoabdominal movement
11.4 Sputum production (quantity, color, consistency, and odor)
11.5 Mental function
11.6 Skin color
11.7 Breath sounds
11.8 Blood pressure
11.9 Pulse oximetry (if hypoxemia with procedure has been previously demonstrated or is suspected); blood gas analysis (if indicated)
11.10 Intracranial pressure (ICP) in patients for whom ICP is of critical importance.

PAP 12.0 FREQUENCY:

12.1 *Critical Care*—from once per hour[43] to once every 6 hours, for intermittent PAP as tolerated. PAP order should be re-evaluated at least every 24 hours based on assessment made during and following each treatment.

12.2 *Acute/Domiciliary Care*

12.2.1 Common strategies for PAP vary from twice to four times daily, with frequency determined by assessment of patient response to therapy.

12.2.2 PAP orders for acute care patients should be reevaluated at least every 72 hours based on patient response to therapy or with any change of patient status.

12.2.3 Domiciliary patients should be re-evaluated periodically and with any change of status.

PAP 13.0 INFECTION CONTROL ISSUES:

13.1 Observe Universal Precautions as appropriate.[44]
13.2 Follow guidelines for prevention of transmission of tuberculosis in health care settings.[45]
13.3 Observe all infection control guidelines posted for specific patient.
13.4 Disinfect any reusable equipment (according to manufacturer's recommendations) between patients.

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Prolonged Neuromuscular Blockade

Hugh S Mathewson MD

Myoneural blocking agents of the nondepolarizer class are essential adjuncts to the management of patients on mechanical ventilators. The major purposes of their use are (1) to achieve patient-ventilator synchrony, (2) to provide restraint in restless or uncooperative patients, and (3) to minimize respiratory work and oxygen consumption in patients with serious cardiopulmonary impairment. Although such agents have been used virtually since the introduction of mechanical ventilators, few guidelines have been offered in the respiratory care literature.

Two recent surveys[1,2] have established that there are wide differences among institutional practices concerning the indications for neuromuscular blockade, choices of agents, dosage regimens, monitoring protocols, and limitations on the use of this method of treatment. A report just published has issued a further warning—that myoneural blocking drugs are being used more often, with a consequent rise in the incidence of prolonged paralysis.[3]

In 1990-1991, a national survey was conducted to assess the current use of sedating drugs and neuromuscular blocking agents in patients requiring mechanical ventilation.[1] Survey forms were sent to head nurses of medical intensive care units (ICUs) in hospitals that had pulmonary fellowship training programs. Of the 164 responding hospitals, nearly all (161) employed neuromuscular blockers. Most of them (115) reported occasional use (on less than 20% of ventilator patients), but 43 reported frequent use, and 3 reported routine use. Pancuronium, given by intermittent intravenous injection, was most frequently the drug of choice (47%), followed by vecuronium (22%) and atracurium (17%). Surprisingly, only 21% of ICUs monitored neuromuscular blockade with peripheral nerve stimulators.

Another survey reported in 1992[2] was directed to anesthesiologist-intensivists, that is, those with the special certificate of competence in critical care from the American Board of Anesthesiology, and had 185 respondents. Vecuronium was listed as the primary neuromuscular blocker by 52%, pancuronium by 28%; only 3% preferred atracurium. The principal reason cited for the selection of vecuronium was its relative lack of cardiovascular side effects. Peripheral nerve stimulators were employed by 34%, monitoring by clinical judgment was reported by 55%, and 11% indicated that no monitoring was used. One intensivist used a servo-controlled computer with a muscle-relaxant infusion titrated to a specific electromyographic response.

A current clinical commentary[3] has indicated that anecdotal reports and small series from more than 30 medical centers have drawn attention to prolonged muscular weakness following the continuous use of a neuromuscular blocking drug for more than 2 days. Recent prospective studies[4-6] and other retrospective series[7-9] suggest that as many as 70% of critically ill patients have extended periods of weakness after prolonged administration of pancuronium or vecuronium. Two principal syndromes have been recognized.

The first syndrome is simply a prolongation of neuromuscular blockade. Altered pharmacokinetics of pancuronium and vecuronium are mainly responsible. Elimination of pancuronium is largely dependent upon renal excretion (about 70%), the remainder being mostly biotransformed and excreted into the bile. In contrast, vecuronium, which is a close chemical congener of pancuronium, mainly undergoes hepatic metabolism and biliary excretion (80%), with only 10-20% renal excretion. Renal impairment can cause marked retention of pancuronium and, to a lesser degree, of vecuronium. Deacetylation of both drugs occurs, mainly in the liver, but the deacetyl derivatives both have appreciable paralyzant activity. The major proportion of reported cases of prolonged blockade are in patients with multiple-organ failure secondary to critical illness, particularly sepsis.[10] Deterioration of liver and kidney function causes demonstrable accumulation of both drug and active metabolite, in plasma[4,11] and in muscle samples.[11]

The second syndrome is an acute myopathy that is manifested when neuromuscular transmission is regained.[12] Muscle wasting occurs, es-

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pecially in the extremities. Creatine kinase levels are often elevated, although this is not a constant finding. Many patients who develop myopathy are asthmatics receiving large maintenance doses of corticosteroids. Douglass and colleagues[5] found that plasma levels of creatine kinase were elevated in 19 of 25 patients mechanically ventilated for severe asthma. All were on high-dose steroids and had received vecuronium continuously for more than 2 days. Nine of these patients developed myopathy. Evidence has accumulated to show that denervated muscle is particularly vulnerable to steroid myopathy. Neuromuscular blocking agents may create the denervated state predisposing to this condition.[13] It is of interest that pancuronium and vecuronium incorporate a steroidal nucleus, although they do not exhibit hormonal side actions.[14] Whether their structural similarities to glucocorticoids contribute to their untoward interactions is unknown.

It may be pertinent to note that paralysis lasting more than 12 hours has not been reported following prolonged administration of atracurium. This is a shorter-acting nondepolarizing blocker that is almost totally biotransformed in the plasma, by ester hydrolysis and Hofmann degradation. Thus, its duration of action is, theoretically, little affected by hepatic or renal disease. Furthermore, its chemical structure is nonsteroidal. One of its biotransformation products is laudanosine, a central stimulant that can produce convulsions in animals but this effect has not appeared in man. Considering the limited ICU experience with atracurium,[15] and with its even shorter-acting congener, mivacurium, no conclusions can definitely be drawn concerning its safety for prolonged neuromuscular blockade.

It is evident that neuromuscular blocking agents will be increasingly required to facilitate newer modes of mechanical ventilation, such as ratio reversal, and for alternative methods of gas exchange, such as permissive hypercapnia and extracorporeal membrane oxygenation. A consensus should be reached on the criteria that determine the indications for continuous neuromuscular blockade. Patients with serious hepatic or renal disease should be given pancuronium or vecuronium with extreme caution, perhaps with serial determinations of plasma levels. These two drugs should not be given for more than 48 hours to any patient receiving sizable doses of corticosteroids.

Monitoring of the degree of paralysis should include assessment by peripheral nerve stimulation and observation of muscle response.[16] Nondepolarizing agents diminish the amplitude of single muscle twitches. A series of 4 supramaximal stimuli at 2/second has been described by Ali and Savarese[17] and is called the train-of-four (T4). The responses have been correlated with the approximate proportion of muscle receptor sites occupied by the blocking drug. If only one twitch is elicited during T4, approximately 90-95% of receptors are occupied; three twitches correspond to 75-80% occupancy, which is usually satisfactory for ventilator control. The return of all 4 twitches is an objective indicator for redosing. The test is helpful in maintaining a steady state and preventing cumulative overdosage. It should be remembered that redosing of nondepolarizers requires much less of the initial dose (perhaps 25%), particularly with pancuronium. If shorter-acting drugs like atracurium are used, it may be preferable to employ a continuous-drip infusion, titrating the rate of administration with the peripheral nerve stimulator.

There are many reasons why continuous administration of a neuromuscular blocking drug should be limited to 2 days or less if possible. Sedative-hypnotic drugs must be given concomitantly to prevent patient awareness and anxiety, thus interfering with sensorial and neurologic evaluation. Alterations in gas distribution occur secondary to diaphragmatic paralysis, and the cough mechanism is eliminated. Also, there is the ever-present risk of catastrophic asphyxia if there is ventilator disconnection or malfunction. The potential of prolonged neuromuscular weakness should be a foremost consideration, together with the realization that there is likely to be increased respiratory morbidity if ventilator dependence is extended. The risks of continuous myoneural blockade require that it be used only on definite indication and with constant reliable surveillance.

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Robert S Campbell RRT and Richard D Branson RRT

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Ventilator-Specific Pressure-Support Algorithms

Despite its relatively uninspiring debut in 1981 as a technique to overcome the imposed work of breathing due to poorly designed demand valves, pressure support ventilation (PSV) has become perhaps the most prominent mode of ventilatory support for the 1990s. PSV has been shown to improve patient-ventilator synchrony, reduce the work of breathing, decrease dead-space-to-tidal-volume ratio, and prevent respiratory muscle fatigue.[1-13] PSV is also unique in its ability to provide partial or full ventilatory support. Microprocessor control provides PSV this flexibility and is also responsible for the array of algorithms by which PSV is delivered. Outwardly, all PSV may seem similar, but, as the quotation suggests, underneath lies unappreciated complexity. This Kittredge's Corner is written to inform practitioners of the phase variables essential to the delivery of a PSV breath and each manufacturer's specific PSV algorithm.

A PSV breath is, by definition, patient-triggered, pressure-limited, and flow-cycled. Each PSV breath thus has four distinct components that may be manipulated or controlled by the ventilator: (1) the trigger sensitivity (each PSV breath may be either pressure- or flow-triggered); (2) the rise-time to PSV level (often referred to as the initial flow or flow gain); (3) the PSV level (which may be set as a change in airway pressure or as a peak airway pressure); and (4) the cycle variable (the primary cycle variable is flow, which is usually either a fixed terminal flow or a percentage of the peak inspiratory flow). A number of 'safety' cycling variables are available on many ventilators that utilize either volume, time, or pressure to terminate inspiration. These safety variables may become active in the presence of a large air leak, patient-ventilator dyssynchrony, or inappropriate ventilator settings.

Until recently, the trigger variable was limited to airway pressure, and the trigger sensitivity was set at the most sensitive level that avoided autocycling. In addition to the sensitivity setting chosen by the practitioner, two other variables may affect the ease of triggering. First, it has been shown that the patient's imposed work of breathing may be dramatically affected by the pressure-sensing site alone and that imposed work may be decreased by moving the pressure-sensing site as close to the trachea as possible.[14] Any resistive element placed between the patient and the pressure-sensing site adversely affects triggering [eg, a heat and moisture exchanger (HME) placed between the endotracheal tube and the Y piece, a heated humidifier on a ventilator that senses pressure within the ventilator on the inspiratory side, or an expiratory filter on a ventilator sensing pressure within the machine on the

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The information provided here was taken from the respective operator manuals. A product manager for each manufacturer was contacted when necessary to provide additional information and to verify the content. Each manufacturer has reviewed the content relevant to its ventilators. The authors have no financial interest in any of the products described.

expiratory side]. Second, the response time of each ventilator's pressure transducer and lag time between the actual pressure detection and delivery of flow to the airway may significantly differ among ventilators.[15] Many ventilators incorporate flow-triggering during PSV. The advantages of flow-triggering PSV breaths are a reduction in lag time compared to pressure-triggering and the presence of a continuous bias flow delivered through the breathing circuit. This bias flow provides gas to the patient at the onset of inspiration, when demand for flow is high. The initial flow supplied by the ventilator is determined by patient effort, the ventilator's PSV algorithm, the PSV setting, and the peak flow capability of the ventilator. Choosing the appropriate PSV algorithm can be important in certain groups of patients[16,17] and has even spawned two conferences specifically addressing the issue of PSV breath delivery.[18,19] Altering rise-time or flow during PSV has been shown to improve patient-ventilator synchrony.[16,17] Our group[16] and MacIntyre and Ho[17] demonstrated that inspiratory flow in excess of patient demand can cause premature cycling, whereas insufficient inspiratory flow results in increased work of breathing. These findings have recently been confirmed in a third study.[20] Only a few ventilators provide for rise-time adjustment.

Although the definition of a PSV breath is consistent among ventilators, each ventilator incorporates a specific PSV algorithm that may differ markedly from others; thus, it is important for practitioners to have a thorough understanding of the algorithm and the design characteristics that affect PSV on specific ventilators (Table 1).

BEAR 3

PSV is a standard feature on the BEAR 3 in the SIMV (synchronized intermittent mandatory ventilation) and CPAP (continuous positive airway pressure) modes. This ventilator has a master PSV ON/OFF switch located on the control panel and a separate, uncalibrated PSV control knob that is used to adjust the PSV level from 5.5 to 66 cm H_2O above baseline pressure. When the master PSV control is in the ON position in the SIMV or CPAP mode, a light-emitting diode (LED) on the display panel alerts the user that the feature is operational. If the control is left in the ON position in the Control or Assist-Control modes, the PSV LED will illuminate intermittently, denoting that the feature is not active but will become active immediately if the mode is changed to SIMV or CPAP. The level of PSV delivered to the patient is displayed on the pressure gauge. Depression of the AIRWAY/SUPPORT button (located below the pressure gauge) displays the Pressure Support Reference pressure, which is typically within 10% of the actual PSV level delivered to the patient. Each PSV breath must be pressure-triggered from the proximal airway, and the sensitivity is set with the uncalibrated ASSIST knob that is adjustable from –1.1 cm H_2O at 2 L/min to –8.0 cm H_2O at 70 L/min. The rise-time to the pressure support level is not adjustable, and the peak flow available to the patient is 100 L/min. During delivery of a PSV breath, the demand valve servo-controls flow to the patient in an attempt to balance measured proximal pressure and set reference pressure. The exhalation valve balloon is inflated by a compensator in order to attain the desired PSV level in a manner similar to that used to maintain the desired PEEP level. Each PSV breath is flow-cycled when inspiratory flow decreases to 25% of peak flow. A PSV breath may also be pressure-cycled if system pressure exceeds the normal pressure limit set by the clinician.

BEAR 5

PSV is a standard feature on the BEAR 5 ventilator in the SIMV, AMV, and CPAP modes of ventilation. The PSV level is set and manipulated using the PRESS SUPP control located on the control panel, and is adjustable from 0 to 72 cm H_2O above the preset baseline pressure. The peak inspiratory pressure during a PSV breath is equal to set PSV level plus the set baseline (PEEP) level. The maximum peak airway pressure attainable for a PSV breath is 72 cm H_2O. When a PSV breath is delivered to the patient, the word Supported appears on the INSPIRATORY SOURCE indicator of the monitor. The CONTINUOUS FLOW control must be set to 0 before PSV can be activated. If the continuous flow is set between 5 and 40 L/min, PSV is automatically turned off. Each PSV breath is pressure-triggered from the proximal airway when the inspiratory pressure drop exceeds the ASSIST SEN-

Table 1. Characteristics of PSV Breaths as Provided by Each Ventilator Manufacturer

Ventilator	Trigger Variable		Adjustable	PSV Range	Flow-Cycle	Set PSV =	Alternate Cycling Criteria			Backup
	Pressure H_2O	Flow	Rise-Time	(cm H_2O)	Criteria	PIP or ΔP	Time (s)	Pressure	Volume	Ventilation ?
Adult Star 1500/2000	−0.5 to −10*	–	–	0 to 70	4 L/min	ΔP	3.5	3.0 cm H_2O above PSV	–	Yes
Amadeus	−0.5 to −10†	–	–	0 to 100	25% of PF	PIP	3.0	High pressure limit	–	Yes
BEAR 3	−1.1 to −8*	–	–	5.5 to 66	25% of PF	ΔP	–	High pressure limit	–	No
BEAR 5	−0.5 to −5*	–	–	0 to 72	20 to 27% of PF‡	ΔP	3.0	High pressure limit	–	No
BEAR 1000	−0.2 to −5*	–	18 position control‡	0 to 65‡ 0 to 80‡	25% of PF	ΔP	5.0	High pressure limit	–	Yes
Evita	−0.2*§	25 mL at 1.5 L/min for 50 s‖	0 to 2.5 s	0 to 80	25% of PF	PIP	3.0	3.0 cm H_2O above PSV	–	Yes
Veolar	−0.5 to −10†	3 to 15 L/min*	–	0 to 100	25% of PF‡	PIP	3.0	High pressure limit	–	Yes
VIP	−1.0 to −20*	–	–	0 to 50	5 to 25% of PF‡	ΔP	0.1 to 3.0 adj or 2 × cycle time	High pressure limit	1.5 L‡	Yes
Wave	−0.1 to −5*	–	–	0 to 60	PSV cycling flow = $(PF)^{0.6}$ (inspiratory time) K‡	ΔP	3.0	2.0 cm H_2O above PSV	4.0 L	No
300	−0.1 to −17‖	0.6 to 2.0 L/min‖	0 to 10% of cycle time	0 to 100	5% of PF	ΔP	> 80% of cycle time	20 cm H_2O above PSV, high pressure limit	–	Yes
900C	−0.1 to −20‖	–	Yes#	0 to 100	25% of PF	ΔP	> 80% of cycle time	3.0 cm H_2O above PSV	–	No
2200B	−0.5 to −5*	–	–	0 to 50	25% of PF, or 4 L/min, whichever is greater	ΔP	–	High pressure limit	–	No
7200ae	−0.5 to −20‖	3 to 15 L/min‖	–	0 to 70	5 L/min	ΔP	3.0	1.5 cm H_2O above PSV	–	Yes
6400/8400ST	−1 to −20*	–	–	0 to 50	25% of PF	ΔP	3.0	–	–	Yes

*Proximal airway.
†Inspiratory outlet.
‡See text.
§Not adjustable.
‖Expiratory outlet.
#By adjusting working pressure.
PF = peak flow; PIP = peak inspiratory pressure; K = the constant.

SITIVITY setting, which is adjustable from –0.5 to –5.0 cm H_2O. The rise-time to the PSV level is not adjustable, and the peak inspiratory flow available to the patient on a PSV breath is 150 L/min. PSV breaths are normally flow-cycled when the inspiratory flow decreases to 20-27% of the peak flow required to achieve the PSV level. The terminal-flow algorithm used by the BEAR 5 attempts to maximize volume delivery and minimize expiratory work of breathing during PSV.

Pressure Support (cm H_2O)	Termination Flow (% of peak)
1- 9	27
10-19	26
20-29	25
30-39	24
40-49	23
50-59	22
60-69	21
70-72	20

Inspiration may be pressure-cycled if the peak inspiratory pressure exceeds the peak Normal-Pressure-alarm setting, or time-cycled if inspiratory time exceeds 3.0 seconds. The minimum setting for the Low-Inspiratory-Pressure alarm is 5.0 cm H_2O; and, thus, when PEEP/CPAP levels of 0 to 1 cm H_2O are used with PSV at < 2 cm H_2O, activation of the Low-Inspiratory-Pressure alarm may ensue.

BEAR 1000

PSV is a standard feature on the BEAR 1000 in the SIMV/CPAP mode. The PSV level is set and manipulated by depressing the PRES SUP/INSP PRES keypad, rotating the control knob to the desired level, and depressing the PRES SUP/INSP PRES keypad a second time to verify the change. The PSV level ranges from 0 to 65 cm H_2O or 0 to 80 cm H_2O, depending on the particular ventilator model used. The PRES SUP/INSP PRES control is a multifunction control that sets the PSV level in the SIMV/CPAP mode, the inspiratory pressure of a pressure-controlled breath, and the minimum inspiratory pressure delivered on a volume-controlled breath when pressure augmentation is activated. Each PSV breath is pressure-triggered from the proximal airway when patient effort results in a change in airway pressure exceeding the ASSIST SENSITIVITY, which is adjustable from –0.2 to –5.0 cm H_2O. The rise-time to the PSV level is clinician-adjustable using the PRESSURE SLOPE control. The PRESSURE-SLOPE setting ranges (using arbitrary numbers) from P –9 to P +9 to –9 to +9, where +9 correlates to the highest initial flowrate and P –9 (P stands for pediatric) corresponds to the lowest flowrate. P –0 and 0 are the neutral default settings for pediatric and adult patients, respectively. PSV breaths are normally flow-cycled when the inspiratory flow decreases to 25% of the peak flow necessary to achieve the set PSV level. PSV breaths may also be time-cycled if inspiratory time exceeds 5 seconds.

Bird 6400/8400ST

PSV is a standard feature on the 6400/8400ST, and the control knob is located on the front panel of the ventilator. PSV is active in the SIMV and CPAP modes and is adjustable from 0 to 50 cm H_2O above baseline pressure. The PSV control sets the pressure level above the PEEP/CPAP setting, and thus peak airway pressure = PSV level + PEEP. When PEEP changes are made, the PSV level (change in pressure) is maintained. PSV breaths are pressure-triggered from the proximal airway, and the sensitivity is adjustable from –1.0 to –20 cm H_2O. Inspiration is flow-cycled when inspiratory flow decays to 25% of peak inspiratory flow. The rise-time to PSV level is not adjustable, and the peak inspiratory flow available during a PSV breath is 120 L/min. The 6400/8400ST incorporates a preset 3-second inspiratory-time limit for all PSV breaths. The number on the PSV control panel flashes when any breath is time-limited. An Apnea-Interval alarm may be set from 10 to 60 seconds, and an apnea backup rate may be set independent of the mandatory breath rate.

Bird VIP

PSV is a standard feature on the VIP ventilator in the SIMV/CPAP mode. The PSV level is set and manipulated using the PRESSURE SUPPORT knob located on the control panel. The PSV level is adjustable from 0 to 50 cm H_2O above the PEEP/CPAP level, and peak inspiratory pressure is equal to the set PSV level plus the PEEP/CPAP level. All PSV breaths are pressure-triggered from the proximal

airway, and sensitivity is adjustable from –1.0 to –20 cm H_2O. Rise-time to the PSV level is not adjustable, and the peak flow available during a PSV breath is 100 L/min. The VIP incorporates specific breath-termination criteria for all PSV breaths in an attempt to minimize premature termination in patients with low compliance and high resistance. The flow-cycling algorithm automatically adjusts the percentage of peak flow at which inspiration is terminated, based on the delivered tidal volume (V_T).

Delivered V_T	Termination Criterion (% of peak flow)
0-50 mL	5
50-200 mL	5-25*
> 200 mL	25

*Percentage increases linearly with delivered V_T.

The VIP also incorporates a variable inspiratory time backup for PSV breaths that is clinician-adjust-able from 0.1 to 3.0 seconds and is set by the INSPIRATORY TIME control. If a leak prevents the flow from decreasing to 25% of the peak flow, the PSV breath will be terminated based on the INSPIRATORY TIME control setting or at twice the cycle time, whichever comes first. If this occurs, the PSV display flashes to indicate that the breath is time- rather than flow-cycled. As a safety feature, after 1.5 L has been delivered, the inspiratory flow decreases to 40 L/min for the remainder of the inspiratory time. PSV breaths may also be pressure-cycled if inspiratory pressure exceeds the High-Pressure Limit set by the clinician.

Dräger Evita

PSV is a standard feature on the Evita in the SIMV, MMV (mandatory minute ventilation), and SPONT (spontaneous) modes of ventilation. The PSV level may be set from 0 to 80 cm H_2O and is independent of baseline pressure; thus, peak airway pressure equals the PSV Control setting. The PSV Control is actually a dual-function knob located in the lower right corner of the control panel. In the CMV mode, this knob controls the "intermittent PEEP," but in any mode allowing spontaneous ventilation the PSV control knob controls the PSV level. Sensitivity is not adjustable and is actually a combination of pressure-, volume-, and flow-triggering in order to avoid auto-cycling. The demand valve has a fixed, preset sensitivity of –0.2 cm H_2O. A pressure-supported breath begins when the demand flow generator has delivered a volume of at least 25 mL and has reached a minimum flow of 1.5 L/min for 50 ms. The rise-time to the PSV level is adjustable using the PRESSURE SUPPORT FLOW knob located in the upper right corner of the control panel. The maximum flow setting (full counter-clockwise) provides a rapid rise to PSV level, whereas the minimum (full clockwise) setting provides rise to the PSV level in approximately 2.5 seconds. Inspiration is flow-cycled when inspiratory flow decays to 25% of the peak flow. Inspiration may also be pressure-cycled if airway pressure exceeds 3.0 cm H_2O above the PSV setting, or time-cycled if inspiratory time exceeds 3 seconds. Backup ventilation is available at a user-selected rate (f) and V_T. The apnea interval is adjustable from 15 to 60 seconds.

Hamilton Amadeus

PSV is a standard feature on the Amadeus in the SIMV and SPONT modes. The PSV control is part of a dual-function control for PEEP/CPAP and Pressure Support located on the control panel. The control knob adjusts the PSV level from 0 to 100 cm H_2O, independent of the PEEP/CPAP setting; thus, peak pressure equals the PSV setting. PSV breaths are pressure-triggered from a pressure transducer located inside the ventilator on the inspiratory side. The sensitivity is adjustable from –0.5 to –10.0 cm H_2O. The rise-time to the PSV level is not adjustable, and the maximum flow possible during a PSV breath is 188 L/min. Inspiration is flow-cycled when inspiratory flow decays to 25% of peak flow. Inspiration may be time-cycled if inspiratory time exceeds 3 seconds or pressure-cycled if airway pressure exceeds the high-pressure limit setting.

Hamilton Veolar

PSV is a standard feature on the Veolarin the SIMV, MMV, and SPONT modes of ventilation. The PSV control is part of a dual-function control

for PEEP/CPAP and Pressure Support located at the lower level of the control panel. The control knob adjusts the PSV level from 0 to 100 cm H_2O independent of the PEEP/CPAP setting; thus, the PSV setting is equal to the peak airway pressure of each supported breath (not PEEP-compensated). PSV breaths may be pressure-triggered from a transducer located in the ventilator on the inspiratory side (adjustable from –0.5 to –10 cm H_2O) or with the Veolar FT upgraded software it can be flow-triggered from a variable-orifice flow-sensor located at the proximal airway. The flow sensitivity is adjustable from 3 to 15 L/min. The maximum flow available to the patient during a PSV breath is 188 L/min. Each PSV breath is flow-cycled when inspiratory flow decreases to 25% of peak inspiratory flow. When using the upgraded software, the clinician may choose the termination-flow criterion by flipping a toggle switch located on the SPECIAL FUNCTIONS panel on the lower right-hand corner of the machine. The default flow-cycle setting for PSV is 25%, and one of the following may be programmed as an option internally: 12.5, 18.75, 32, or 37.5%. PSV breaths may also be pressure-cycled if airway pressure exceeds the high-pressure limit alarm or time-cycled if inspiratory time exceeds 3.0 seconds. Backup ventilation is available at a user-selected f and V_T. The apnea interval may be set at 20 or 40 seconds.

Infrasonics Adult Star 1500/2000

Positive pressure support (PPS) is a standard feature on the Adult Star ventilator that is functional in the pressure- or volume-controlled SIMV and CPAP modes of ventilation. PPS is set and manipulated on the Ventilator Settings Screen #1 or the Graphics Monitoring Screen #3 by rotating the SELECT knob until the cursor aligns with POS PRES SUP, depressing the ENTER button to access the PPS level, rotating the SELECT knob to change the PPS level to the desired setting, and depressing the ENTER button again to verify the change. The PPS setting is adjustable from 0 to 70 cm H_2O above baseline pressure. The PPS setting actually sets the change in pressure above baseline, and thus peak pressure equals PPS + PEEP/CPAP level. PPS breaths are pressure-triggered at the proximal airway, and the sensitivity is adjustable from –0.5 to –10.0 cm H_2O. The PPS breaths are normally flow-cycled when inspiratory flow decays to a preset flowrate of 4 L/min. Rise-time to PPS setting is not adjustable, and the peak flow available to the patient during PPS is 160 L/min. PPS breaths may be pressure-cycled if inspiratory pressure exceeds the PPS setting by 3 cm H_2O, and may be time-cycled if inspiratory time exceeds 3.5 seconds.

Newport Wave

PSV is a standard feature on the Wave ventilator in the SIMV (pressure- or volume-controlled) and spontaneous modes of ventilation. PSV is set and manipulated using the PSV knob on the front control panel, and is adjustable from 0 to 60 cm H_2O above baseline PEEP setting. All PSV breaths are pressure-triggered from the proximal airway. Sensitivity is adjustable from –0.1 to –5.0 cm H_2O. Bias flow for spontaneous breaths is also available and is adjustable in conjunction with PSV. The range for bias flow is 0 to 30 L/min, and it must be set properly in order to stabilize baseline pressure and decrease the response time of the demand valve. The manufacturer recommends that a bias flow of 5 L/min be used in conjunction with PSV to counter the problems associated with endotracheal tube leaks. It should be noted that the patient's inspiratory flow demand must exceed the bias flow in order to meet the sensitivity requirement to initiate a PSV breath. The peak flow available to the patient during a PSV breath is 150 L/min. The rise-time to PSV level is machine-controlled using "predictive learning," which uses feedback from flow and proximal-pressure sensors. The rise-time is selected by the microprocessor by choosing one of five flow settings delivered from the servo-controlled valve in an effort to (1) minimize the period of time that airway pressure is below baseline and (2) eliminate premature cycling of inspiration and pressure overshoot. Each PSV breath is flow-cycled when

$$\text{PSV cycling flow} = (\text{peak flow})^{0.6}\,(\text{inspiratory time})\,K,$$

where K = the constant.

The table that follows gives examples of terminal-flow criteria for PSV breaths based on the peak inspiratory flow and inspiratory time.

Peak Flow (L/min)	Inspiratory Time (seconds)					
	0	0.5	1.0	1.5	2.0	2.5
10	3.5	6.3	9.1	—	—	—
30	7.0	12.5	18.0	23.5	29.0	—
50	9.7	17.2	24.7	32.3	39.8	47.4
70	11.9	21.2	30.5	39.8	49.1	58.4
90	13.9	24.8	35.7	46.5	57.4	68.3
110	15.8	28.1	40.4	52.7	66.9	79.5
130	17.5	31.2	44.8	58.5	72.2	85.9
150	19.1	34.1	49.0	64.0	78.9	93.9

A PSV breath may also be volume-cycled if delivered V_T exceeds 4.0 L, pressure-cycled if the airway pressure exceeds 2.0 cm H_2O above the set PSV level, or time-cycled if inspiratory time exceeds 3.0 seconds.

Puritan-Bennett 7200 Series

PSV is a standard feature of the 7200 series. The PSV control is located in the SPECIAL FUNCTIONS menu, which is accessed by pressing the "++" key on the control panel. PSV is function #10 on the menu. To enter or change the PSV level, the ENTER button must be pressed while the menu cursor is highlighting #10. The appropriate numeric keypad should then be used to enter the desired PSV level and the ENTER button depressed twice in order to activate the new PSV level. The PSV level is adjustable from 0 to 70 cm H_2O and is functional in the SIMV and CPAP modes. The PSV setting is the actual change in pressure above baseline PEEP/CPAP; thus, the peak inspiratory pressure on spontaneous breaths is equal to the PSV level plus the set PEEP. PSV breaths may be pressure-triggered from a pressure transducer inside the ventilator on the expiratory side, and the sensitivity is adjustable in 0.5-cm-H_2O increments from –0.5 to –20 cm H_2O. PSV breaths may also be flow-triggered if the Flow-By 2.0 option is purchased from the manufacturer ($1,295.00 new, $395.00 as an upgrade to the Flow-By 1.0 version). The flow sensitivity is adjustable from 3 to 15 L/min. The rise-time to the PSV level is not adjustable, and the peak flow capability during a PSV breath is 180 L/min (120 L/min if the compressor is on). The cycle variable of PSV breaths is a nonadjustable, fixed flow of 5 L/min. That is, when inspiratory flow decays to 5 L/min, the inspiratory phase is terminated, and the ventilator cycles into exhalation. Inspiration may be pressure-cycled if airway pressure exceeds the PSV level by 1.5 cm H_2O. A PSV breath may also be time-cycled if inspiratory time exceeds 3 seconds.

Apnea ventilation is available at user-selected f, V_T, and F_{IO_2}. The apnea interval is adjustable from 20 to 120 seconds.

Sechrist 2200B

PSV is a standard feature on the 2200B ventilator that is available in the SIMV, MMV, and CPAP modes of ventilation. The PSV level is set and manipulated using a calibrated control knob termed PRESSURE LEVEL, which is located on the front panel. This knob also sets the pressure level attained during a pressure-controlled breath, thus preventing the use of a separate PSV level when providing pressure-controlled breaths in modes that allow spontaneous breathing. The PSV level is adjustable from 0 to 50 cm H_2O above baseline pressure; thus, the peak inspiratory pressure is equal to the PSV setting + PEEP. All PSV breaths are pressure-triggered from the proximal airway, and the trigger sensitivity is adjustable from –0.5 to –5.0 cm H_2O. The rise-time to the PSV level is not adjustable, and the peak flow available to the patient is 120 L/min on a PSV breath. PSV breaths are flow-cycled when inspiratory flow decreases to 25% of peak flow, or 4 L/min whichever is greater. PSV breaths may also be pressure-cycled if airway pressure exceeds the set Peak-Pressure-Limit alarm.

Siemens 300

PSV is a standard feature on the 300 ventilator in the SIMV (pressure control) + Pressure Support, SIMV (volume control) + Pressure Support, and Pressure Support modes. PSV breaths may be flow-triggered or pressure- and flow-triggered. The flow sensitivity is adjustable from 0.6 to 2.0 L/min (adult range) by rotating the TRIG SENSITIVITY knob within the uncalibrated green area. For combined flow-pressure triggering, the control should

be set between 0 and −17 cm H_2O. During combined triggering, inspiration is initiated when the patient's effort results in an inspiratory flow greater than the continuous flow plus a decrease in expiratory pressure corresponding to the control knob setting. PSV is adjustable from 0 to 100 cm H_2O above PEEP (which is adjustable from 0 to 50 cm H_2O) using the PRESSURE SUPPORT LEVEL ABOVE PEEP knob located on the left side of the control panel. Peak pressure during a PSV breath is equal to PSV setting + PEEP. The PSV knob has safety catches at 30, 60, and 80 cm H_2O and must be manually manipulated in order to set PSV above each level. The rise-time to PSV level is adjustable from 0 to 10% of the cycle time using the INSP RISE TIME % knob. When this is set at 0, pressure increases to its preset level at the start of inspiration. The maximum flow attainable during a PSV breath is 180 L/min. A safety catch is located at the 1% position on the control knob. Inspiration is flow-cycled when inspiratory flow decays to 5% of the peak inspiratory flow during that breath. Inspiration may also be time-cycled if the inspiratory time exceeds 80% of the set cycle time, or pressure-cycled if (1) measured airway pressure exceeds the preset PRESSURE CONTROL LEVEL ABOVE PEEP by 20 cm H_2O, or (2) the Upper-Pressure-Limit alarm is reached.

A ventilator-set and adjustable-pressure support-type breath is delivered in the Volume Support mode of ventilation, and will be discussed in detail in a Kittredge's Corner dedicated to microprocessor-controlled closed-loop ventilation.

Siemens 900C

PSV is a standard feature on the 900C in the SIMV + Press Support and Press Support modes. The PSV level is adjustable from 0 to 100 cm H_2O and is set and manipulated using the tNSP PRESS LEVEL ABOVE PEEP control knob. A safety catch is located at 30 cm H_2O. The peak inspiratory pressure equals the inspiratory pressure level set plus the PEEP level. All PSV breaths are pressure-triggered from a transducer located inside the ventilator on the expiratory side. The sensitivity is adjustable from 0 to −20 cm H_2O. The rise-time to the PSV level may be indirectly set by adjusting the working pressure on the ventilator. Each PSV breath is flow-cycled when inspiratory flow decreases to 25% of the peak inspiratory flowrate. Inspiration may also be pressure-cycled if airway pressure exceeds the PSV level by more than 3 cm H_2O, or time-cycled if inspiratory time reaches 80% of the preset respiratory cycle time (by-product of the respiratory rate setting).

Discussion

PSV has been a standard feature incorporated into the design of every adult critical care ventilator designed in the last decade. However, no consistency exists among manufacturers' approaches to delivering PSV breaths. This paper is not intended to comparatively evaluate the ventilators' PSV programs, but rather to describe how each functions and to list the characteristics of each. Clinicians should be aware of the differences in PSV delivery and familiar with the subtleties of the ventilators they use routinely.

The indications, clinical application, titration, and weaning of PSV have been described at length elsewhere and readers are referred to the literature for further review.[1-13,21] This Kittredge's Corner is dedicated to discussing the essential, desirable (optional), and unnecessary characteristics of a ventilator's PSV program in regard to the four phase variables, user interface, and safety concerns (alarm systems).

We believe that PSV should be included as a standard feature on every new ventilator manufactured. The PSV control should be clearly marked and easily accessible to the user. The PSV status (ON/OFF) and level should be clearly and continuously displayed. The PSV control should be disabled in modes that do not allow spontaneous breathing. When the ventilator is changed to a spontaneous breathing mode, the ventilator should alert the clinician that PSV is active and should require the user to verify the set PSV level.

The Trigger Variable

We believe that flow-triggering is superior to pressure-triggering because it is associated with a lower patient work of breathing (WOB) to initiate a PSV breath. Flow-triggering systems should have an adjustable flow sensitivity in order to avoid ventilator auto-cycling in the presence of leaks in the

system. The bias flow need not be clinician-adjustable, although in a small percentage of patients this may be advantageous. Flow-triggering systems that decrease the bias flow during the initial period of exhalation may decrease expiratory WOB, resistance, and, possibly, the auto-PEEP level. Flow-triggering systems should also be unaffected by humidifying devices or the development of condensation in the breathing circuit. Flow-triggering systems should have a pressure trigger available as a backup. The operator should be able to discontinue flow-triggering temporarily for various clinical reasons (eg, metabolic measurements or delivery of medications to the airway). The ventilator's volume and flow measurements should be unaffected by or compensated for the extra flow necessary to provide flow-triggering.

Pressure-triggering is certainly acceptable as a means of initiating a PSV breath, but clinicians must understand the mechanics involved. Ventilators that monitor pressure in the proximal airway are preferable to those monitoring pressure within the ventilator, on either the inspiratory or expiratory side. It has been shown that pressure-triggering from the distal tip of the endotracheal tube (trachea) is comparable to flow-triggering inspiration, in a model of spontaneous breathing.[14] At the present time (May 1993), tracheal pressure monitoring and triggering are not available from any ventilator manufacturer. Pressure sensitivity should always be set to the most sensitive setting that avoids ventilator auto-cycling.

The Rise-Time to PSV Level

Originally, the rise-time to the PSV level was preset by the manufacturer and/or limited by the maximum flow capabilities of the particular ventilator. Recently, it has been shown that proper adjustment of the rise-time to PSV level decreases the patient's WOB and improves patient-ventilator synchrony.[16,17] Because a major advantage of PSV is patient control of inspiratory time, V_T, flowrate, and f, allowing these variables to be manipulated by the clinician may have both positive and negative results. We strongly believe that any ventilator that allows the practitioner control over the rise-time should also provide a graphics package that can display both pressure and flow vs time and pressure-volume loops, to assist the user in attaining the proper setting. A properly set rise-time results in an initial flowrate that is low enough to avoid overshooting the PSV level, yet high enough to reach the PSV level in the early part of inspiration, when patient effort is highest. Figure 1 shows examples of pressure and flow waveforms that result from various rise-time settings. For the adjustable rise-time feature to be used successfully, respiratory care practitioners must understand what this feature controls and what the resultant effects are. The rise-time control affects the initial flow delivered into the breathing circuit from the ventilator and adjusts the attack angle of the pressure waveform from very aggressive (vertical waveform) to passive (sinusoidal pressure rise). We believe that graphic interpretation of PSV breaths is essential for properly setting and titrating the rise-time. Achieving a square pressure waveform should be the goal when one adjusts the rise-time control. It must be realized that the rise-time setting also has a secondary effect on the cycling of a PSV breath. Ventilators that use a percentage of the peak inspiratory flow to cycle may be affected in two ways as a by-product of the rise-time setting. First,

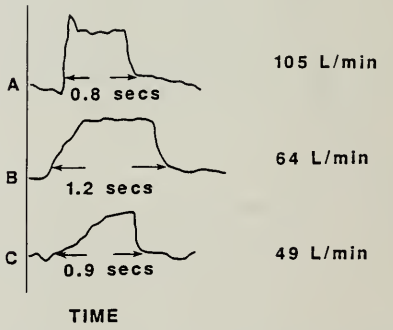

Fig. 1. Effect of changing inspiratory rise-time in a patient preferring a midrange flow. In A, flow is in excess of patient demand and the pressure spike is seen. In B, as flowrate is decreased, inspiratory time (t_i) lengthens and the pressure spike is absent. Machine output matches patient demand. When flowrate is further reduced (C), patient demand exceeds machine flowrate and t_i falls. Deformation of the pressure waveform during the rise to the set PSV level is also seen in C. (Reprinted, with permission, from Reference 16.)

as the initial flow of a PSV breath increases, so does the flow at which cycling occurs. Second, when the initial flow of a PSV breath exceeds patient demand for flow or when airway resistance is high, the flow profile becomes steeper (ie, flow decelerates more rapidly). Example: On a ventilator that uses 25% of peak flow as the cycling variable, when the peak flow is 50 L/min, the cycling flow is 12.5 L/min. If by increasing the rise-time, initial flow is increased to 75 L/min, the flow-cycle point becomes 19 L/min.

Improper setting of the rise-time may cause premature cycling of a PSV breath. As mentioned, high initial flow results in both an increased flow-cycle point and a faster deceleration to terminal flow. If patient demand is 60 L/min, the flow cycling occurs at 15 L/min (25% of peak flow). If flow is increased to 90 L/min, the cycling occurs at 22.5 L/min. This may cause inspiration to end while the patient continues to inspire. When PSV breaths are prematurely cycled, an inadequate tidal volume may be delivered, as evidenced by tachypnea and increased patient WOB. Premature cycling of a PSV breath may also occur if the initial flow is set too low. When the flow is set too low, patient effort ceases before the PSV level is reached. This also results in inadequate tidal volume delivery and tachypnea.

The rise-time control should have a neutral default or initial setting, and the control setting should be continuously visible to the practitioner.

The Pressure Support Level

The PSV level should be adjustable in 1-cm-H_2O increments to a minimum of 50 cm H_2O above the PEEP level. The PSV level should be PEEP-com-pensated—which means that when PEEP changes are made, the change in airway pressure on all PSV breaths is maintained. Use of dual-function controls may confuse practitioners, and we encourage manufacturers to incorporate a dedicated PSV control into the design of future ventilators.

Many current ventilators allow the PSV level to change without human interaction to achieve some user-selected end point such as V_T or minute volume (V_E). Two ventilators (BEAR 1000 and Bird 6400/8400ST) allow the PSV level to exceed the set level to deliver a guaranteed V_T on every PSV breath. The user selects the minimum V_T and flow-rate that are clinically acceptable for the patient. As flow decays to the minimum setting, if the minimum V_T has not been delivered, that minimum flowrate is maintained and the inspiratory time is lengthened until the set V_T is delivered. Figure 2 shows an example of what happens to flow, volume, and pressure when this approach is employed.

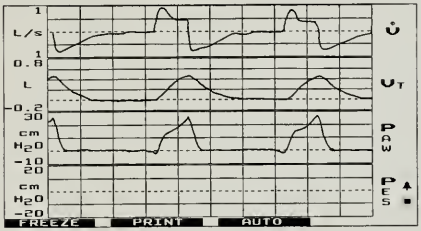

Fig. 2. Pressure, flow, and volume waveforms that result from a volume-guaranteed PSV breath delivery. This technique allows pressure to increase above the PSV setting in order to deliver a minimum V_T on each breath.

Two other ventilators allow the PSV level to change on a breath-by-breath basis to meet a user-selected end point of V_E (Hamilton Veolar) or V_T (Siemens 300). In the MMV mode, the Veolar predicts patient V_E from the average f and V_T of the last 8 breaths delivered. If the predicted V_E exceeds the user-selected MMV, then the ventilator decreases the PSV level in 1-3 cm H_2O decrements to a user-selected baseline level. If the predicted V_E is less than the MMV setting, then the ventilator autoregulates the PSV level up in 1-3 cm H_2O increments to the maximum value or until the high-pressure limit is met. It is essential to properly set all alarms (especially high f, apnea interval, and high-pressure limit) for immediate notification of changes in patient status. In the Pressure Regulated-Volume Controlled mode of ventilation, the Siemens 300 makes breath-to-breath calculations and pressure adjustments to deliver the user-selected V_T. When this mode is initiated, the ventilator gives the patient 3 test breaths at 10 cm H_2O and calculates system compliance based on the volume delivery. The ventilator then gives the next breath at 75% of the pressure needed (based on sys-

tem compliance) to deliver the set V_T. From this point on, the ventilator updates the compliance measurement breath by breath and adjusts the pressure level in order to achieve the set V_T.

The described ventilator-driven PSV programs are designed to give the patient and the caregiver 'the best of both worlds.' In the initial part of inspiration, the patient receives flow that is responsive to his/her demand, which improves patient-ventilator synchrony and is associated with a decreased WOB. In the middle of the PSV breath, the patient can still control the volume, flow, and flow decay from the ventilator, which adds to patient comfort. At the end of each PSV breath, the patient is guaranteed a minimum V_T delivery, which adds to patient safety. Practitioners are reminded that when using these PSV programs it is essential that all alarms and controls be set appropriately so that any change in patient status will be recognized quickly and easily.

The Flow-Cycle (Terminal-Flow Criterion)

The terminal-flow criterion for terminating a PSV breath was initially determined by the manufacturer based on lung-model studies. Although this variable is perhaps the most important one in regard to PSV breath delivery, results of controlled clinical trials evaluating different terminal-flow criteria have not been published. Terminal flow can be characterized as (1) a preset percentage of the peak flow delivery; (2) a preset level of terminal flow; (3) a percentage of the peak flow delivery that is machine-adjusted based on either inspiratory time, PSV setting, or V_T delivery; or (4) a percentage of peak flow that is user-adjustable. Published clinical trials suggest that differences in terminal-flow criteria are clinically unimportant. It must be noted that at the time of these studies the rise-time to PSV level was not adjustable. We believe that it may be advantageous to have some control over the cycle variable when manipulating rise-time in order to prevent premature termination of the inspiratory phase. Further work is needed to determine the optimal flow-cycling criterion.

We believe that the preset level of terminal flow is the least desirable flow-cycling mechanism. Most PSV breaths delivered from ventilators using this criterion actually are pressure-cycled rather than flow-cycled. Figure 3 depicts the flow, volume, and pressure waveforms of a PSV breath delivered from a 7200 series ventilator that is pressure-cycled. Pressure-cycling of a PSV breath suggests that the patient is attempting to exhale before the flow-cycle criterion is met. In addition, with the recent popularity of noninvasive mechanical ventilatory support (full face mask or nasal mask) using PSV, leaks are to be expected at a level that prevents flow-cycling of the PSV breaths (> 5 L/min). Flow-cycling in this manner would also be unacceptable in the neonatal/pediatric population, which uses uncuffed endotracheal tubes.

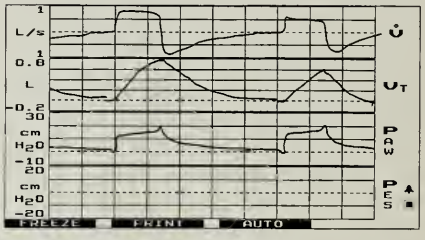

Fig. 3. Pressure and flow waveforms that depict a pressure-cycled PSV breath delivered by a 7200 series ventilator. Note that the inspiratory flowrate does not decelerate to the 5 L/min terminal flow.

Little is known about the ventilator-adjustable percentage of terminal flow, and clinical studies are needed to assess their efficacy.

Alternate Cycle Criteria

As a safety feature, most ventilators provide a backup cycling mechanism. The alternate cycling mechanism must be either pressure, volume, or time. Most of the ventilators have a preset alternate cycling criterion (ie, 2 cm H_2O above PSV setting, 3-second inspiratory time limit). Some ventilators have alternate cycling that can be adjusted by the practitioner. The high-pressure limit is common to most ventilators for alternate pressure-cycling, whereas the Siemens ventilators and the Bird VIP allow manipulation of the maximum inspiratory time as a by-product of the respiratory rate setting (cycle time).

When one of the alternate criteria is met, the ventilator should alert the practitioner with either a visual or audible alarm.

Alarms and Apnea Backup

Essential alarms that must be available and properly set during ventilation with PSV include high f, high inspiratory pressure, and apnea. Optional alarms that would enhance the safety of a PSV program include low V_T, high/low V_E, and low inspiratory pressure.

We believe that it is essential for each ventilator, in addition to detecting apnea, to have an adjustable apnea interval and backup mandatory ventilation when PSV is used in the CPAP/SPONT mode of ventilation.

Summary

PSV is associated with an improvement in patient-ventilator synchrony and decreased patient work of breathing. These improvements are largely the result of the ventilator allowing the patient to control inspiratory flow, V_T, time, and respiratory rate. As the complexity of mechanical ventilators increase, control of important breath variables is placed in the hands of subjective respiratory care practitioners. It is imperative that practitioners understand the indications and effects of varying PSV parameters (the rise-time and cycle criteria) in order to maintain the most efficient and comfortable breath delivery possible.

Eventually, ventilators will have the ability to automatically make adjustments in order to deliver the 'perfect' breath to match patient demand and need. This may be accomplished by allowing the practitioner to program the ventilator to deliver a set waveform (in this case pressure) or the ventilator algorithm may 'independently' decide which waveforms to deliver in order to maximize efficiency. In either case, respiratory care practitioner input and understanding is essential.

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PFT Corner #50— Is It Really Asthma?

Sandra E King AS RRT, Pamela Leisenring AS RRT, and Robert H Warren MD

Case Summary

A 16-year-old Caucasian boy was referred to Arkansas Children's Hospital for evaluation of wheezing, which had been present since age 18 months, and decreased exercise tolerance. Asthma had been diagnosed at age 13 based on volume-time curves and peak flow meter data suggesting obstruction (Table 1, Fig. 1). Post-bronchodilator tracings had not been obtained, but according to the clinical history the patient had been treated aggressively with bronchodilators and steroids with no improvement. Respiratory symptoms progressed during the ensuing 3 years, primarily manifested by increased exercise intolerance. The patient had a history of chronic sinusitis and positive skin tests for house dust and mold, and had been treated with immunotherapy for 3 years. Physical examination was normal. Daily medications consisted of cromolyn so-

Table 1. Results of Forced Expiratory Maneuver in 16-Year-Old Boy with Long History of Wheezing and Exercise Intolerance

	Predicted	Actual (% Predicted)
FVC (L)	5.1	5.4 (106)
FEV_1 (L)	4.5	2.5 (55)
FEV_1/FVC (%)	≥ 70	46 (–)
FEF_{25-75} (L/s)	4.5	1.8 (40)
PEFR (L/min)*	580	300 (52)

*Obtained with a Wright peak flow meter.

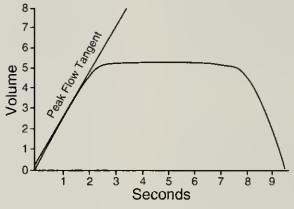

Fig. 1. Volume-time tracing generated by 16-year-old boy with long history of wheezing and exercise intolerance.

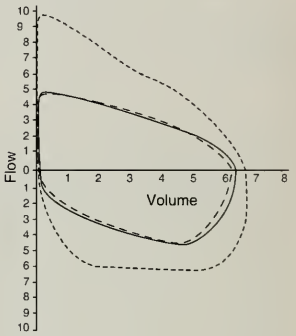

Fig. 2. Flow-volume loops from subject in Figure 1. Predicted is dotted line; before bronchodilator is solid line; after bronchodilator is dashed line. (Also see values in Table 2.)

Table 2. Flow-Volume Loop Values with Pre- and Post-Bronchodilator Study in 16-Year-Old Boy with Long History of Wheezing and Exercise Intolerance

	Before Bronchodilator		After Bronchodilator
Variable	Predicted	Actual (% Predicted)	Actual (% predicted)
FVC (L)	6.62	6.33 (96)	6.17 (93)
FEV_1 (L)	5.40	3.94 (73)	3.94 (73)
FEV_1/FVC (%)	—	63%	64%
FEF_{50} (L/s)	5.93	3.35 (56)	3.56 (60)
FEF_{25-75} (L/s)	6.33	3.18 (50)	3.42 (54)
FEF_{75} (L/s)	3.32	1.93 (58)	2.57 (77)
PEFR (L/s)	9.74	4.73 (49)	4.67 (48)
PIF (L/s)	—	4.67	4.55
FIF_{50} (L/s)	—	3.33	3.48
FEF_{50}/FIF_{50}	—	1.01	1.02

Ms King and Ms Leisenring are Pulmonary Laboratory Therapists, and Dr Warren is Chief of Pulmonary Medicine, Arkansas Children's Hospital, and Professor, Department of Pediatrics, University of Arkansas for Medical Sciences—Little Rock, Arkansas.

This case summary was presented by Ms King during the RESPIRATORY CARE OPEN FORUM at the 1992 AARC Annual Meeting held in San Antonio, Texas.

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dium (Intal) and albuterol aerosol three times each day and 5 puffs of flunisolide (Aerobid) twice daily. Prior to exercise, albuterol (Proventil rotacaps) and cromolyn by metered dose inhaler were used. Flow-volume values obtained at our institution before and after bronchodilator are shown in Table 2; the tracings appear in Figure 2.

Fig. 3. Focal narrowing of the esophagus as seen on fluoroscopy after barium swallow.

Questions

1. How do you interpret the volume-time curve and the peak flow values?
2. How do you interpret the flow-volume-loop data?
3. Was there a positive response to bronchodilator?
4. Does inspection of the flow-volume loop lead you to any conclusions?
5. Are additional tests needed? If so, what do you recommend?

Answers and Discussion on Next Page

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Answers and Discussion

1. Interpretation of PFT: The volume-time curve shows a normal FVC (106% of predicted) with a reduced FEV_1 (55% of predicted). The FEV_1/FVC of 46% and the FEF_{25-75} of 40% of predicted indicate airflow limitation. The peak expiratory flow-rate (PEFR) of 51% of predicted is reduced. The volume-time curve is nonspecific for location of airflow limitation.

2. Interpretation of Flow-Volume Loop: The flow-volume-loop data show a normal FVC of 96% of predicted with a reduced FEV_1 (73% of predicted). The FEV_1/FVC of 63% is reduced, indicating airflow limitation. The results suggest obstruction both in the large airways with a PEFR of 49% and in the small airways with a FEF_{25-75} of 50% of predicted. The PIF and the FIF_{50} are similarly reduced.

3. Response to Bronchodilator: The flow-volume loops pre- and post-bronchodilator reveal no response to aerosolized isoproterenol.

4. Inspection of Flow-Volume Loop: Inspection of the flow-volume curve is essential for interpretation. In a normal configuration, the initial forced expiratory maneuver creates the maximum peak of the flow-volume curve. This is the effort-dependent portion of the curve and represents primarily large airway function. The effort-independent portion of the flow-volume loop follows in a decreasing linear fashion and is a result of the elastic recoil of the lung. This portion of the curve represents small airway function. Inspection of this patient's curve reveals a flattened pattern of the middle and/or early portion of the maneuver in both inspiration and expiration—a pattern consistent with fixed upper-airway obstruction. In this type of obstruction, inspiratory and expiratory flows are generally decreased in about equal proportions because the lesion is unaffected by intrathoracic pressure changes.[1] This patient's configuration, combined with lack of response to bronchodilator, further suggests fixed upper-airway obstruction. Inspection of the flow-volume loop leads one to consider a diagnosis of severe tracheal malacia, tracheal stenosis, tumor, or vascular ring.

5. Further Testing: Further evaluation was needed to diagnose the obstruction. Barium swallow remains the best single diagnostic technique for patients with complete vascular ring. Bronchoscopy can be utilized to confirm the location and severity of extrinsic airway obstruction but is neither essential nor necessary in all patients because a diagnosis can usually be made without bronchoscopy.[2,3] In this patient, a barium swallow and fluoroscopy (Fig. 3) revealed focal narrowing of the esophagus at the level of a right-sided aortic arch. A smaller left-sided aortic arch was present at a slightly inferior level. There was a 50% narrowing in the lateral tracheal dimension (Fig. 4). Subsequent cardiac catheterization confirmed a diagnosis of vascular ring with tracheal compression. The patient underwent a left thoracotomy for surgical repair of his vascular ring, without complications.

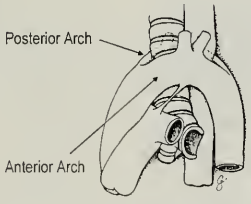

Fig. 4. Vascular ring comprised of a posterior and anterior arch encircling the trachea. (Reprinted from Reference 7, with permission.)

Discussion

A double aortic arch is the most common anomaly of the aortic arch causing significant respiratory and swallowing symptoms.[4] Aortic arch anomalies resulting in tracheoesophageal compression were recognized in 1794 by Beyford.[5] A clear understanding of normal aortic arch development is necessary to comprehend the abnormal development that leads to these arch anomalies. The six primitive aortic arches first arise from the mesoderm at 3 weeks of life, and then regress leaving segments of the III, IV, and VI ventral and dorsal roots that evolve into the mature anatomy of the aortic arch, the arch vessels, pulmonary artery, and ductus arteri-osus.[6] Abnormal development or inappropriate persistence of segments of the primitive arches results in an abnormal configuration of the mediastinal vessels.[7] Affected patients may have recurrent respiratory infections. Older children may have a history of wheezing, which has been treated for months or years as asthma.[2] Surgery does not always reverse the tracheal narrowing, but most patients become asymptomatic. Since surgery, this patient has had no stridor or breathing difficulties at rest. However, no important improvement can be seen with forced spirometric maneuvers. Our patient's chief complaint is his inability to tolerate competitive sports. His exercise limitation is a result of the persistent airway narrowing in an area of focal tracheal malacia, caused by the vascular ring.

In Conclusion

Although volume-time curves suggest airflow limitation, they did not provide information for specifically determining a fixed airway obstruction. In our patient, the shape of the flow-volume loop with no response to bronchodilator strongly suggested

fixed obstruction and led to a correct diagnosis. Analysis of the flow-volume loop is a component of the evaluation of wheezing and exercise intolerance.

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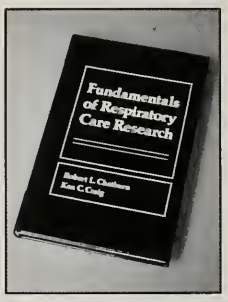

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RETROSPECTROSCOPE REDUX:

EITHER—OR? (Why Not Both?)

• Humphrey Davy in 1800 prepared his own hydrogen, put a measured amount of it in a spirometer, breathed it back and forth 7 times, and then, from the initial and final concentrations of H_2 in the spirometer, measured his own lung volume.

• John Hutchinson in 1840 measured vital capacity and its subdivisions in several thousand normal subjects, proposed normal values for men and women of different height, weight, and age, and devised a test to measure maximal voluntary inspiratory and expiratory effort. He also used his tests to diagnose pulmonary disease before it could be detected by other means.

• Luigi Luciani in 1877 estimated intrapleural pressure in animals from measurements of their intraesophageal pressure, obtained from a balloon-catheter placed in the esophagus.

• Marie Krogh in 1915 published details of her single-breath carbon monoxide test to measure pulmonary diffusing capacity in man.

• Hürter in 1912 punctured the radial artery of man to obtain blood samples, and Stadie in 1919 used this technic to measure arterial O_2 saturation of patients with pneumonia. Van Slyke and Neill developed their manometric method for measuring blood O_2 and CO_2 in 1924.

• Fleisch devised his meter for measuring instantaneous rate of air flow in 1925.

Reprinted with permission, from the American Review of Respiratory Disease 1975;111:539-542.

Despite these pioneering studies stretching back 140 years, in 1940 only a few clinical physiologists in a few countries—and practically no physicians—had an interest in measuring pulmonary function in man. Indeed, in 1940 no textbook of physiology included even a paragraph on pulmonary function (apart from vital capacity). But only six years later the golden age of clinical pulmonary physiology began. What happened between 1940 and 1946? These, of course, were the years in which we first prepared for and then participated in World War II. But we had also participated in World War I in 1917-1918 and no special benefits accrued to pulmonary physiology.

Why "yes" in 1940-46 and "no" in 1917-18? I believe several factors were responsible. President Roosevelt decided in 1940 to organize a National Defense Research Committee (NDRC) to coordinate research in developing military weapons—followed in July 1941 by a second decision, to form an Office of Scientific Research and Development (OSRD). The OSRD (headed by Vannevar Bush) had two divisions—the existing NDRC and a new Committee on Medical Research (headed by an eminent renal physiologist, A N Richards, later to become President of the National Academy of Sciences). Roosevelt, Bush, and Richards believed that solution of certain physiologic problems would help the over-all war effort and that their solution required *(1)* support of biomedical research by government (Army, Navy, Air Force, and the new OSRD); *(2)* full use of government and university laboratories; *(3)* the building of new research facilities for both government and universities when needed;

(4) the recruiting of well-trained scientists, in and out of uniform, to work in them, with at least some freedom to investigate basic as well as targeted problems.

Another important factor was that the time was ripe for the solution of certain physiologic problems. What were these problems and who worked on them? The Air Force had fighter planes with open cockpits (pressurized cabins weighed more and reduced speed and maneuverability). It was easier to increase pressure only in the pilot's alveoli than in the whole cabin—but no one knew whether pilots could tolerate increased alveolar pressure and, if they did, whether it would improve oxygenation of their arterial blood and permit them to climb to higher altitudes and gain tactical advantage over enemy planes. The OSRD and then the Air Force asked Wallace Fenn to work on pressure breathing, and soon the team of Fenn, Otis, and Rahn, working at the University of Rochester, had completed their classic studies on the pressure-volume relations of the lungs and thorax and the work of breathing, and the modern era of pulmonary mechanics had begun. Pressure breathing was not new in 1942 but Fenn, Otis, and Rahn *were* new, both to respiratory physiology and to human physiology, and fresh minds looking at an old problem made a big difference.

The Air Force had another problem—how to identify when and where inward leaks of air occurred in oxygen masks when a fighter pilot moved his head quickly up and down and side to side. John Lilly, in his first postdoctoral year in Detlev Bronk's Johnson Foundation for Medical Physics at the University of Pennsylvania drew on his engineering background at California Institute of Technology to devise an instrument (now known as a nitrogen meter or analyzer) that recorded continuously and almost instantaneously the nitrogen concentration in an extremely small sample of air. This instrument a few years later became the keystone in single-breath analysis of uneven distribution of inspired air to alveoli and is now widely used to measure "closing" volume. Leak detection was not new in 1942, but John Lilly was and again it made a big difference.

The Air Force also needed to know how fully arterial blood was saturated with oxygen. One way was to draw arterial blood and analyze it. But, in 1942, arterial puncture was far from an accepted procedure. The needs of aviation medicine widened its use by clinical investigators, and then, as fears about its dangers faded, they introduced it into clinical medicine. I did my first arterial puncture on Bob Dripps in 1942 and, since he survived, he then did his first on me. At the end of the war, it became a standard procedure in pulmonary function laboratories.

But the Air Force also wanted to measure arterial O_2 saturation *continuously* in subjects during simulated flight conditions. Glenn Millikan was ready with the answer. Millikan had worked in Barcroft's laboratory at Cambridge in the 1930s, had used the Hartridge-Roughton continuous flow apparatus (for measuring rapid chemical reactions) to study the velocity of the O_2-myoglobin reaction, and had modified this apparatus to make it rugged, compact, and portable. Millikan joined the staff of the Johnson Foundation in 1939, about the time Germany went to war with England. Because of his strong ties with England, Millikan wanted to do something to help in its war effort. He knew that it would be important to pilots to know their arterial O_2 saturation (he himself was a mountain climber and his father-in-law, Mallory, was an early climber of Mt Everest); toward this end he developed the first ear oximeter. It was widely used in aviation laboratories in wartime and then, with improvements, in physiologic investigation in peacetime.

Riley and Lilienthal began their classic studies on pulmonary diffusing capacity for O_2 (D_{O_2}) while they were assigned to the Pensacola Naval Air Station. They wondered why normal subjects at high altitude became more cyanotic during exercise and specifically whether the deepening cyanosis was caused by failure of P_{O_2} in blood and gas to come to near-equilibrium when alveolar P_{O_2} was low. Although their D_{O_2} method is not widely used now, it reopened the field of pulmonary diffusing capacity and led to their own comprehensive examination of ventilation-blood flow relationships in the lung.

At the University of Southern California low pressure chamber, two physiologists, Drs Scott and Drury, were studying the effects on man of different types of pressure breathing—increased pressure only during inspiration, only during expiration, or during both. This required separating the inspiratory and expiratory lines; the simplest way was to use a large-bore 3-way aluminum stopcock and turn it manually from the inspiratory to the expiratory line during each breath. One day an engineer visited their laboratory. The physiologists wondered if this tedious manual task could be done by electrically operated valves. The engineer thought the task could be done better with a mechanical valve. His name was Ray Bennett; in a short time, he developed the first "flow-sensitive" (now called "Bennett") valve, the forerunner of many sub-

sequent automatically controlled valves for intermittent positive pressure breathing.

Linus Pauling wrote in 1946, "On October 3, 1940, at a meeting in Washington, called by Division B of the National Defense Research Committee, mention was made of the need for an instrument which could measure and indicate the partial pressure of oxygen in a gas. During the next few days we devised and constructed a simple and effective instrument for this purpose." This was the Pauling oxygen meter that capitalized on existing knowledge that the magnetic susceptibility of oxygen was considerably greater than that of other gases.

The Medical Division of the Chemical Corps at Edgewood, Maryland, also had biologic problems to solve. One was to determine the cause of death of occupants of nearly closed, poorly ventilated pill boxes when flame throwers were used o clear them out. Was it heat alone? Or anoxia (due to exhaustion of O_2 by flame)? Or increased concentrations of CO or CO_2 (caused by incomplete or complete combustion)? The answer required portable, rapidly responding gas analyzers to measure gas concentrations continuously within the pill boxes. Infrared absorption spectra of gases were well known and Fastie and Pfund had already constructed infrared analyzers. These were quickly modified for field use. Pulmonary physiologists were there and remembered to use them constructively when peace came.

Edgewood arsenal also housed pilot plants that manufactured war gases such as phosgene. Occasional accidents there exposed workers to inhalation of phosgene. Some of these workers developed tachypnea and dyspnea and Morton Galdston and I were assigned the job of measuring their lung function. The first tests of pulmonary function that I participated in were on these workers in a laboratory at nearby Johns Hopkins Hospital. This led directly toward my later interest both in pulmonary function and in the causes of dyspnea.

Cardiac catheterization had been used in man long before 1940. Forssmann had catheterized his own heart in 1929 and radiologists had used the technic in the 1930s to inject radiopaque materials in the right heart to improve pulmonary arteriography. And in 1941 Cournand, with Ranges, had written about right arterial catheterization to obtain samples of mixed venous blood. But it was the investigation of traumatic shock in man in wartime that brought together Cournand, Riley, Gregerson, Bradley, and Richards (among others) to study a large number of patients by many technics, one of which was measurement of right atrial pressure. World War II did not discover either arterial puncture or cardiac catheterization, but it did much to speed their acceptance into clinical investigation and then into clinical medicine in the post-war period.

I'm sure that other new devices and concepts originated in the 1940-46 period or that their development or acceptance was speeded during this time. I've mentioned some that I believe redirected a number of scientists into new types of research once the war was over—and whose new activities then paid off handsomely in pulmonary physiology. One example in my own laboratory was Ward Fowler, my first post-war, postdoctoral fellow in 1946. I asked him what he wanted to study. He thought a bit and answered that almost everyone was measuring physiologic changes *dis*continuously and that he'd like to measure something continuously. I replied along these lines: "Here's Millikan's ear oximeter that estimates arterial O_2 saturation continuously, and Lilly's meter that measures the N_2 concentration of a gas continuously and a flow meter that measures air flow continuously." Fowler put them together and ushered in the era of rapid, single-breath analysis of pulmonary function.

Something that had no direct connection with pulmonary physiology also helped to keep the new crop of scientists working on lungs and respiration and to attract many more—and that something was the new science of chemotherapy, greatly accelerated by the needs of wartime. When chemotherapy of tuberculosis arrived in 1946 and when antibiotics could be counted on to keep alive patients who formerly succumbed to a wide variety of chronic pulmonary diseases, pulmonary medicine became a different kind of specialty with broad interests in the diagnosis, prevention, and management of all types of pulmonary diseases and respiratory problems. At last it became a challenging specialty to those who previously had passed it by.

This look into the retrospectroscope gives us a glimpse into the golden era of *applied* pulmonary physiology. Does this glimpse argue for the support of applied, mission-oriented, targeted research? Indeed it does! And this brings us back to the title of this note: "EITHER—OR?" I have little patience with basic scientists who argue that only fundamental research is worthy of support and equally little patience with engineers and clinicians who argue that mission-oriented research is all that we need. This is the EITHER one OR the other approach—one that in a few years predictably leads to a marked decline in the quality of science. The BOTH approach makes more sense, although it raises more

problems. The BOTH approach requires that someone must decide *how much of each.* And someone must decide *if, when,* and *why* support for one should be increased and *for how long* without detriment to the other. It requires genius to answer these questions. Someone had it in World War II; who but a genius (or a lucky gambler) could have assigned Fenn (who had never done research on man), Rahn (a youngster investigating why rattlesnakes changed color), and Otis (another youngster interested in grasshopper eggs and in the oyster heart) to work on pressure breathing in man and have it pay off? Not all applied research pays off; someone less than a genius had assigned Fenn in World War *I* to study the effect of changing the hydrogen ion concentration on the viscosity of dough! The recent, very costly space program has widely publicized its "spin-off" for science, but it had no demonstrable effect on pulmonary medicine. It produced a bit of miniaturization here and there but little else. Among the highly touted accomplishments' of the space program were telemetry and the mass spectrometer. But Einthoven had written a paper on telemetry in 1907, 60 years before the space age, and pulmonary physiologists were using mass spectrometers in the early 1950s (borrowed from researchers in the oil industry who used them long before then).

World War II put good scientists into intimate contact with fascinating problems ready for solution and their enthusiasm carried over into new, rewarding post-war careers. And it had a tremendous spin-off—its success led in a few years to a new type of peacetime research organization—the National Institutes of Health!

JULIUS H COMROE JR

Books, Films Tapes, & Software

Progressive Health Care Management Strategies, by Donald N Lombardi PhD. Soft-cover, 371 pages, illustrated. Chicago: American Hospital Publishing Inc, 1992. $39.95 for AHA members, $49.95 for nonmembers of AHA.

According to the preface, **Progressive Health Care Management Strategies** is intended to be a handbook for the health care manager. In Part I, the author examines the role of the effective health care manager and provides dozens of strategies that can be utilized by managers at any level of the organization. There are six chapters in Part I: The Need for Progressive Strategies; The Organizational Leadership Imperative; Value-Driven Management; Progressive Motivational Strategies; Management of Conflict, Change, and Cultural Diversity; and Critical Line Management Strategies. Each chapter describes situations that might be encountered by the manager and gives reasonable methods for handling them. For example, in Chapter 5, "Management of Conflict, Change, and Cultural Diversity," the author defines conflict; explains causes of conflict and why conflict is difficult to deal with; and, in a very practical fashion, addresses how to deal with conflict. During this time of change in the medical field, I find the chapter dealing with crisis management especially interesting.

Part II of the book, The Progressive Health Care Organization, presents not only line-management but also organization-wide aspects of progressive health care management. The author uses up-to-date examples and graphs, tables, and organizational charts to provide an overview of issues related to line managers, "human resource strategies," and "human resource management." It also includes examples of performance evaluations, orientation flow sheets, and performance measurement worksheets. The chapter entitled "Progressive Business Strategies" makes the reader aware of the progressive business tactics that are used by many first-rate hospitals, thereby allowing the reader to consider application of such tactics in his/her setting.

The last part of the book contains five appendixes that I find to be most practical. Included in these worksheets are outstanding examples that can be used by managers as they evaluate employees' performance. Appendix A includes 20 examples of "organizational values" that can be used to evaluate employees and 100 questions for interviewing prospective employees. Appendix B presents a model performance evaluation that includes duties, responsibilities, goals, and objectives. The author walks the reader through an example of a job description and includes ideas to help motivate the employee to gear his performance appropriately toward compensation. The final appendix includes progressive motivational strategies. Utilizing worksheets allows the manager to work with the employee to help identify motivators for himself/herself.

The first two thirds of the book is well written, with outstanding practical applications that allow the reader to more fully understand the points being made. Although I have heard a lot of the material before, the book is written in such a way that I find it motivating. The book is one that takes concentration and time to complete, and therefore it moved slowly for me. However, all points were well worth reading and offered many new insights.

The appendixes moved quickly as most pages required just a glance and the thought, Should I or How can I utilize this type of worksheet? I strongly recommend this book as worthwhile, even for the seasoned manager.

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All You Really Need To Know To Interpret Arterial Blood Gases, by Lawrence Martin MD FACP FCCP. Soft-cover, 225 pages, illustrated. Philadelphia: Lea & Febiger, 1992. $19.95.

Dr Martin (who is Chief, Division of Pulmonary and Critical Care Medicine, Mt Sinai Medical Center, Cleveland, and Associate Professor of Medicine, Case Western Reserve University School of Medicine) wrote this book to provide the reader with the necessary skills to interpret blood gases in the clinical setting. The author's teaching approach is one of instruction and practice through the use of numerous examples, questions, and practice problems; in fact, the author cautions the reader, "Don't read this book without a pencil in hand." The answers to questions are provided either immediately (in the next paragraph of the text) or at the end of the chapter.

The book is divided into 10 chapters, four of which deal with the relationship between P_{aCO_2} and alveolar ventilation, P_{aO_2} and $P_{(A-a)O_2}$, S_{aO_2} and oxygen content, and the relationships among pH, P_{aCO_2}, HCO_3^-, and acid-base status. These four chapters provide the bulk of the information needed to do a thorough

interpretation of arterial blood gases. The remaining chapters deal with various case studies requiring the integration of information gained in earlier chapters. The P_{aCO_2} equation, alveolar gas equation, oxygen content equation, and the Henderson-Hasselbalch equation are presented in succession, with a more than adequate number of practice problems for each. The clinical application of each equation is presented in a logical and interesting manner, again using the question-and-practice-problem format to reinforce concepts.

The author quickly leads one to a mastery level that focuses on thorough review and consideration of reported 'blood gas' values. In addition to the usual interpretation of pH, P_{aCO_2}, HCO_3^-, and P_{aO_2}, the integration of serum electrolytes, serum CO_2, and the anion gap into the patient's acid-base status is presented and reinforced through clinical case studies.

The author states in the preface that "this is not a physiology textbook" and assumes that the reader has an understanding of respiratory and cardiovascular physiology. He also assumes that the reader has a basic understanding of serum electrolytes and certain categories of drugs (ie, acetazolamides). This prerequisite knowledge limits the book's usefulness for the entry-level health care professional.

There are a few errors, both typographical and conceptual, that need to be addressed. In Figure 5-1 on Page 70, the reader is told that ". . . 30 of 32 Fe^{++} heme sites are bound with O_2 so the S_{aO_2} is 93.8%." The accompanying illustration shows a representation of eight hemoglobin molecules with the four Fe^{++} heme sites on each available for carrying O_2, with one unoccupied site on each of two of the hemoglobin molecules. The reader is left with the impression that per cent saturation is an expression of the number of sites on hemoglobin occupied by oxygen divided by the total sites available for O_2 carriage. The chemistry of hemoglobin is such, however, that molecules tend to be fully saturated or fully unsaturated, with a very small percentage carrying one to three molecules of oxygen (Dickerson R, Geis I. Hemoglobin: structure, function, evolution, and pathology. Menlo Park CA: The Benjamin/Cummings Publishing Co Inc, 1983:23). Question 5-7 on Page 95 asks "With 100% inspired oxygen, approximately how many atmospheres of pressure are necessary to provide an arterial oxygen content equal to that available when breathing room air at sea level? Calculate your answer for a patient with normal lungs and hemoglobin content." Although the question asks one to calculate oxygen content, the answer to the question only reflects the amount of oxygen that is dissolved and not the amount that is also chemically combined with hemoglobin. Figure 6-1 on Page 113 is an illustration of the four primary acid-base disorders and their compensatory changes utilizing the Henderson-Hasselbalch log ratio, ie,

$$pH \cong \frac{HCO_3^-}{P_{aCO_2}}.$$

A series of arrows is used to indicate the direction of the primary disturbance and its resultant effect on pH. The compensation for the primary disturbance is illustrated in a second column by using smaller arrows to indicate the direction of change in the compensatory component and effect on pH. In the Compensatory Event column, the arrow showing the direction of compensation for respiratory alkalosis is pointing in the wrong direction.

I have the impression that the book is directed toward medical students and would be difficult reading for a first-year respiratory therapy student. I do believe, however, that it is a valuable companion text for anyone studying acid-base physiology or for the library of any practicing clinician. Except for the few errors that I mentioned above, the book is clearly worded, arranged in a logical manner, and does what it purports to do—"To teach you how to interpret and wisely use blood gas values."

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Coloring Guide to Regional Human Anatomy, 2nd edition, by Alan Twietmeyer PhD and Thomas McCracken MS. Soft-cover, illustrated, 214 pages. Philadelphia: Lea & Febiger, 1992. $17.50.

Coloring Guide to Regional Human Anatomy is a workbook designed to actively involve the allied health, nursing, or medical student who is enrolled in a human anatomy course. This second edition includes additional illustrations and text modifications and deletions based on comments from students and instructors who used the first edition. Dr Twietmeyer is Associate Professor and Chairman of the Division of Physical Education at Concordia College in Ann Arbor, Michigan. Mr McCracken is Associate Professor and Director of Biomedical Illustration and Communication in the Department of Anatomy and Neurobiology at Colorado State University in Fort Collins, Colorado.

The guide is prefaced with a section on Latin and Greek prefixes, suffixes, and root words commonly used in the study of gross anatomy. Each alphabetical listing gives the Latin (L) or Greek (G) derivation, the meaning, and an example. There is also a blank line that the student is encouraged to fill in as new anatomic

terminology is added to his/her vocabulary. The material is presented "regionally," in four units—Upper Limb; Thorax, Abdomen, and Pelvis; Head and Neck; and Lower Limb. Each unit contains three or four exercises in an outline format. Illustrations are provided with identifying letters or numbers that correspond to the outline; the authors encourage students to label and color these illustrations as a means to increase active participation and improve information retention. Also many opportunities are provided to list the attachments and innervations of muscles. Each exercise contains a section entitled "For Review and Thought," which allows the student to both review and apply the knowledge gained from that particular exercise. Units conclude with an outline of the circulatory system for the region studied. This logical sequencing, as well as the review sections, should assist students in understanding the relationships among anatomic structures.

The outline format is useful for both student and instructor. Students are exposed to the material in a conceptual framework, and are given access to "just the facts," as opposed to wading through an extensive body of often tedious material. Instructors can utilize the outline format to eliminate sections or exercises that may not be applicable to their course or to highlight exercises and/or sections that they consider of more importance than others.

The illustrations are detailed and precise and include surrounding anatomy, thus illustrating interrelationships among structures. Ample room is provided at the end of each exercise for notes. The index is exhaustive, with good cross-referencing, making it easy for the student to find information. **Coloring Guide to Regional Human Anatomy** is a useful tool for the respiratory therapy student enrolled in a gross human anatomy course (with or without a laboratory component) and a good reference and/or review for allied health or nursing professionals.

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Algorithmic Application of PCV

A few comments are warranted regarding the article "Pressure-Control Ventilation with a Puritan-Bennett 7200a Ventilator: Application of an Algorithm and Results in 14 Patients" published in RESPIRATORY CARE in January 1993.[1] We would like to compliment Mr Howard on a fine article that focuses on clinical research—a direction in which we believe respiratory therapy needs to continue to expand. In addition, we agree that the term *inverse ratio ventilation* (IRV) need not be restricted to drastic I-E ratios. As pointed out by Marcy and Marini,[2] one of the benefits of IRV is to increase mean airway pressure; in this context, perhaps a better term for IRV is *extended ratio ventilation.*

Three points need to be addressed in the algorithm. (1) If tidal volume (V_T) increases after a patient is changed from volume-controlled decelerating flow ventilation (VCDFV) to pressure-controlled ventilation (PCV), the practitioner should decrease the peak airway pressure level (P_{set}). The algorithm shows that the P_{set} should be increased. V_T should rise as P_{set} is increased.[3] (2) The algorithm gives the practitioner three choices if V_T decreases after the switch to PCV. One of the choices is to decrease inspiratory time (t_i). If the overall goal is to keep the original V_T constant, decreasing ti will only increase V_T if auto-PEEP is being generated. This is because auto-PEEP reduces the pressure gradient driving flow into the lung ($P_{set} - P_{alv}$). However, in the transition from VCDFV to PCV, Howard states that "t_i was increased in increments of 0.1 second until exhaled V_T was observed to decrease. The t_i immediately prior to this decrease was then used as the t_i for PCV." In the use of this method of determining t_i no auto-PEEP will be developed; therefore decreasing t_i any further will not increase V_T. With a mathematical model developed by Marini et al[3] and later confirmed by Burke et al,[4] V_T can be shown to be a function of several variables during PCV. In Figure 1, note that V_T during PCV increases only when per cent inspiration (t_i%) decreases from very high values (0.95) to approximately 0.75 (arrow in Fig. 1). In fact, over the clinically useful range of t_i%, V_T is not influenced by t_i% (in ARDS-type conditions). This is because the maximal V_T that can be delivered during PCV is

$$V_T = (P_{set})(C_{rs}),$$

where C_{rs} is the total respiratory system compliance.

At low C_{rs}, maximal volume delivery (because it is small) occurs at a very short absolute t_i. Once the maximal V_T ($P_{set} \times C_{rs}$) is delivered and inspiration has not cycled into expiration, alveolar pressure is held at the P_{set} value without any further volume delivery. In addition, Figure 1 also shows that in other simulated lung conditions changing t_i% can increase or decrease V_T depending on what the original t_i% was. (3) The algorithm suggests increasing t_i% when there is no change in P_{aO_2} following the switch to PCV. Wouldn't this only increase mean airway pressure and possibly expose the patient to auto-PEEP and high mean alveolar pressures? If P_{aO_2} does not fall in the transition to PCV, do any changes need to be made in t_i%?

As a final point, in the discussion Howard correctly points out that the

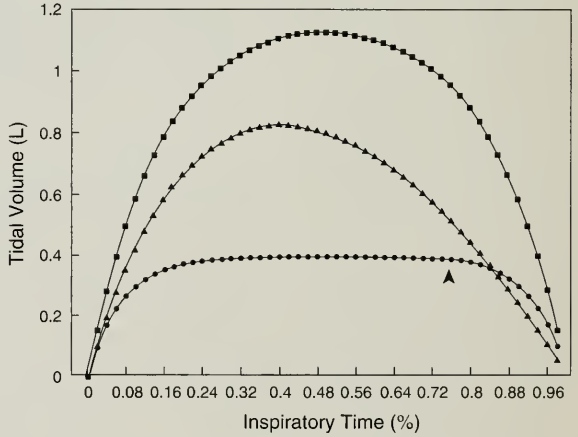

Fig. 1. Data generated using a mathematical model for pressure control ventilation proposed by Marini et al.[3] The settings common to each curve were P_{set} = 20 cm H_2O, and frequency = 15 breaths/min. ARDS curve (-●-) was generated with an inspiratory and expiratory resistance of 15 cm $H_2O \cdot s^{-1} \cdot L^{-1}$ and a C_{rs} of 0.02 L/cm H_2O. COPD curve (-▲-) was generated with an inspiratory resistance of 15 cm $H_2O \cdot s^{-1} \cdot L^{-1}$, an expiratory resistance of 30 cm $H_2O \cdot s^{-1} \cdot L^{-1}$, and a C_{rs} of 0.08 L/cm H_2O. The normal curve (-■-) was generated with an inspiratory and expiratory resistance of 10 cm $H_2O \cdot s^{-1} \cdot L^{-1}$ and a C_{rs} of 0.06 L/cm H_2O.

peak airway pressure limit becomes useless in detecting airway obstruction during PCV and that this fact should make clinicians cautious about using PCV. However, when PCV is used (in adults), airway obstruction will be accompanied by a precipitous fall in V_T. Therefore, it becomes very important for the clinician to set very tight low- and high-exhaled V_T-alarm limits so that changes in resistance (obstruction) and/or compliance will be detected.

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Howard responds:

I agree with the general comments in (1) and (2) of Burke and Van Scoder's letter. However, I warn the reader to be cautious when clinically applying a mathematical model of delivered tidal volume (V_T). The model may be applicable, yet one might have different V_T test results if more clinically realistic values of "P_{set}" typical of ARDS patients are used. ARDS patients can require minute volumes of 20-40 L and experience changing compliance and resistance. One must appreciate and be cautious about bedside application of laboratory theory. Finally, newer ventilators (eg, Adult Star from Infrasonics Inc, San Diego CA) offer adjustable inspiratory flow during PCV.

One can envision and demonstrate test conditions under which delivered V_T is remarkably affected when flowrate is the sole adjustment. In combination with the variables noted earlier, V_T measurements made clinically may differ from measurements made using pure, controlled, mathematical models. Figures 1-3 show the V_T obtained with a Bio-Tek VT-2 test lung under test conditions described in Burke and Van Scoder's letter. Results appear similar at $P_{aw\text{-}peak}$ of 20 cm H_2O (Fig. 1). When higher $P_{aw\text{-}peak}$ values such as those seen in ARDS are used (60 cm H_2O, for example), the effective V_T declines more rapidly at the extreme ends of the t_i% spectrum (Fig. 2). This decrease in V_T is further accentuated at a ventilator rate (f) of 30 breaths/min, not unusual in ARDS. However, in order to support my speculation, these issues should be studied further and reported.

The algorithm, as originally published in January, contained an error.[1] That was unfortunate but has since been corrected in the February issue. Burke and Van Scoder are cor-

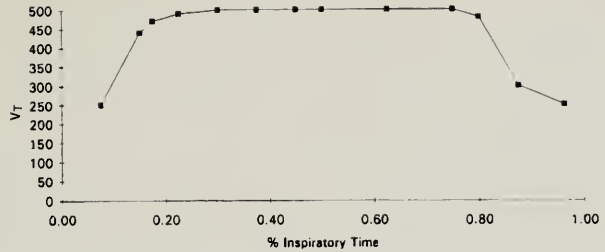

Fig. 1. Changes in tidal volume (V_T) at $P_{aw\text{-}peak}$ 20 cm H_2O, f 15 breaths/min, and different % inspiratory times.

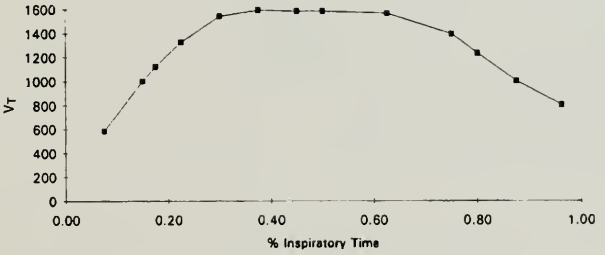

Fig. 2. Changes in tidal volume (V_T) at $P_{aw\text{-}peak}$ 60 cm H_2O, f 15 breaths/min, and different % inspiratory times.

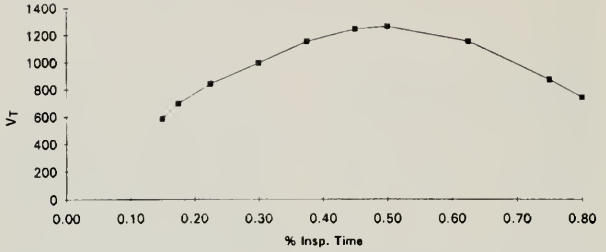

Fig. 3. Changes in tidal volume (V_T) at $P_{aw\text{-}peak}$ 60 cm H_2O, f 30 breaths/min, and different % inspiratory times.

rect in their comments regarding the relationship between inspiratory time and V_T in the presence of auto-PEEP. Regarding Comment 3, please remember we make a transition to PCV by mimicking CMV values. We next ask ourselves, Which direction should we take to 'improve' upon the patient's CMV management?

Burke and Van Scoder ask if lengthening t_i% would expose the patient to auto-PEEP. Unless auto-PEEP exists in CMV from settings incorporated and 'mimicked' during transition, it should not occur if you employ the strategy to lengthen t_i (noted in my paper and verified on Page 2 of their letter: "By using this method of determining t_i, no auto-PEEP . . ."). Of course, one must frequently monitor for auto-PEEP, and I suggest vigilant use of Flow-Time graphics and low exhaled-V_T monitoring to alert the clinician to this problem. As to their follow-up question: "If P_{aO_2} does not fall in the transition to PCV, do any changes need to be made in t_i%?" I can only respond with a question of my own—Why make the transition from CMV if you are not willing to make adjustments differing from the mimicked CMV settings?

The primary purpose of using PCV is to gain independent control of inspiratory time (t_i) and peak airway pressure ($P_{aw\text{-}peak}$). Our efforts are intended to clinically improve a patient's previous clinical situation. Bear in mind that the $P_{aw\text{-}peak}$, F_{IO_2}, and $P_{(A-a)O_2}$ were clinically unacceptable in the patients to whom we applied PCV. We were attempting to maximize control and take advantage of the t_i without compromising (but rather benefiting) the patient. My belief is that with application of t_i as suggested, clinical improvements can occur. I demonstrated this without statistically altering t_i% from CMV levels!

It should be pointed out that the algorithm is used to manage patients after transition as well as at transition. As a result of post-transition manipulation of available ventilator variables (and/or changes in the patient's compliance and resistance), one may be faced with iatrogenic generation of auto-PEEP and subsequent decrease in V_T from baseline. The chart merely serves as *a guideline*, for suggesting options that may be useful in maintaining acceptable V_T. I have found that the chart helps to minimize the effects of operators' having dissimilar skill levels. Ventilator management via algorithm can be very helpful in the environment of the teaching hospital, in which one may encounter a variety of ventilator management styles. Algorithms provide the staff (therapists, residents, nurses, house officers, and medical students) with a method of providing consistent pressure-control ventilator management.

Finally, I appreciate Burke and Van Scoder's last comment on the need to monitor exhaled V_T. This point needs to be emphasized because not all of the manufacturers have heeded this warning. A little pressure from our community may be helpful.

I appreciate Burke and Van Scoder's efforts in responding to the article on PCV. Their input is welcome and the ongoing dialog that they have provided is constructive. The application of PCV is difficult. Without similar communication from many clinicians, the use of PCV may not receive the attention that I think it deserves. We can all learn from this process. Thank you.

William R Howard MBA RRT
Director, Respiratory Care
New England Medical Center
Boston, Massachusetts

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1. Howard WR. Pressure-control ventilation with a Puritan-Bennett 7200a Ventilator: application of an algorithm and results in 14 patients. Respir Care 1993;38:32-40 [Correction to Fig. 1 Respir Care 1993; 38:317].

Reader Enjoys Retrospectroscopes

I am writing to express my appreciation for the reprints of Dr Comroe's Retrospectroscopes in the January and March issues.[1,2] I recall seeing a collection of these in a book with the same title (Von Gehr Press, Menlo Park CA). Of particular interest was a review of the early investigation of retrolental fibroplasia (RLF). I'd really like to see that one again.

My reading of many articles on scientific and medical topics makes

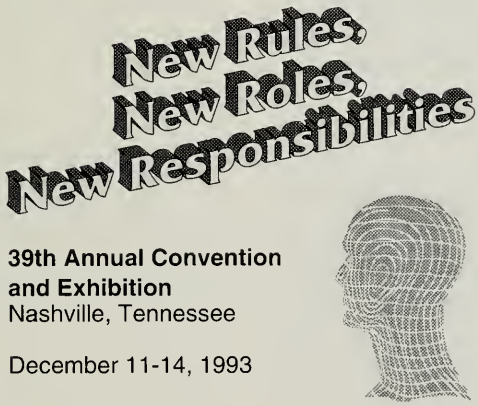
me wonder whether there is a prize for presenting the most exciting information in the dullest possible language. Some authors seem to be aiming for such a prize. By contrast, the intelligence and humor in Comroe's writing still bring a breath of fresh air.

Keith Hopper MA RRT
Medsoft of Vashon
Vashon Island, Washington

REFERENCES

1. Comroe JH Jr. Retrospectroscope 1975—pulmonary diffusing capacity for carbon monoxide (D_{LCO}) (classic reprint). Respir Care 1993; 38:141-143.
2. Comroe JH Jr. Retrospectroscope redux: a sea of air (classic reprint). Respir Care 1993;38:301-302.

Editor's Note: This issue carries another of Dr Comroe's gems, "ETHER-OR? (Why Not Both?)," a history of the measurement of pulmonary function. Future revived Retrospectroscopes will bear such intriguing titles as "Frankenstein, Pickwick, and Ondine," "How To Make Hassenpfeffer," "Inflation—1904 Model," "Hydrogen, Balloons, and Pressures," "What Makes the Sky Blue?" "Tell It Like It Was," "Who's Who in World History," "The Courage To Fail," and "The WITCH Doctor, the WHICH Doctor, and the WISH Doctor." We have been unable to locate the RLF piece Mr Hopper mentioned. Perhaps some kindly reader will help us find it by consulting the book he cited—or checking Retrospectroscopes in issues of the American Review of Respiratory Disease from 1975 on. Information (issue date, page number, perhaps a photocopy of the article) should be sent to RESPIRATORY CARE, *11030 Ables Lane, Dallas TX 75229. Tel 214-243-2272. Fax 214-484-6010.*



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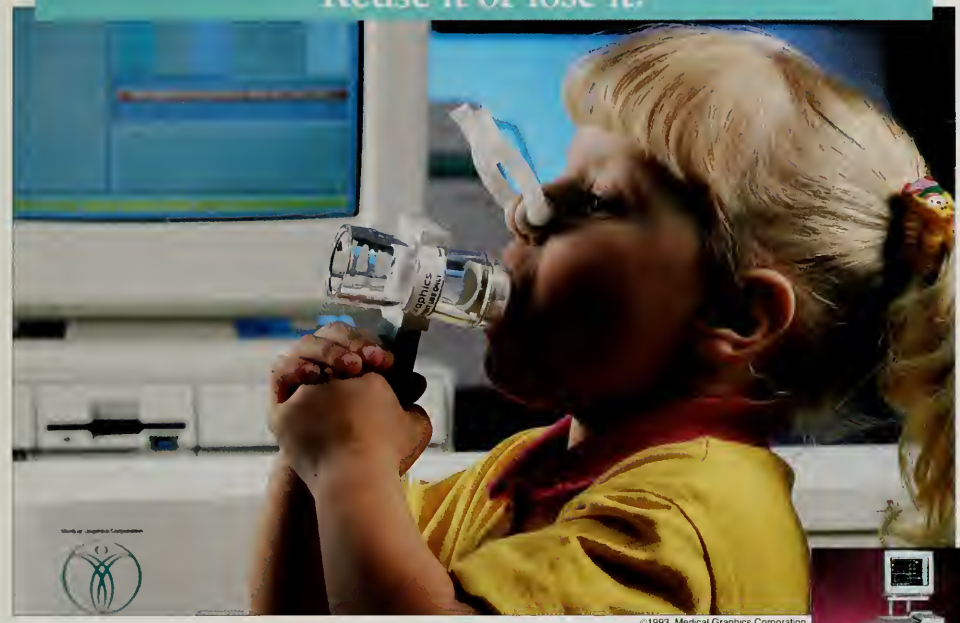

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